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CLOSURE OF OPERATIVE FENESTRAE IN THE LABYRINTH

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Fistulization of the horizontal semicircular canal and production of a fenestra in the labyrinth by covering the artificial opening with some sort of a mobile membrane is an operation which is being performed more and more every day. I am not concerned here with the fact that fistulization produces immediate improvement of hearing in cases of ankylosis of the stapes, nor shall I go into the theories of how this takes place. It suffices to say that immediate improvement occurs when the perilymph is decompressed, and if a sufficiently mobile membrane can be placed over the opening in the labyrinthine capsule the hearing usually remains more or less improved unless something happens to interfere. The greatest difficulty has been that in 2 of every 3 cases the fistula has closed within two months, and in an additional proportion of cases it has closed in six months to a year.[1] If the covering of the fistula becomes bony, whatever initial improvement of hearing may have occurred is lost. It is not understood why some fenestrae close and others remain open. In this paper an attempt is made to clarify some of the factors in this problem; it deals largely with the mechanisms of bone growth in the labyrinthine capsule as they occur in monkeys when an artificial fistula is made in the horizontal semicircular canal and is covered with a skin flap from the membranous meatal wall.

Relatively little has been done in the way of animal experimentation to discover why fistulas become inactive postoperatively. Canfield[2] has been repeatedly cited as showing that there is less regeneration of bone

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1. Fowler, E. P., Jr.: Critique of Surgical Treatments for Deafness, Canad. M. A. J. **43**:546 (Dec.) 1940.

2. Canfield, N.: Labyrinthine Fistulas, Arch. Otolaryng. **30**:62 (July) 1939.

if defects are made in a cat's skull with a dental polishing burr instead of a traditional cutting instrument, such as a chisel or a curet. It has been deduced that fistulization of the labyrinth should therefore be performed with a dental burr rather than with a hand cutting instrument to reduce the incidence of bony closure. Unfortunately, Canfield's observations were made on fistulas in the membrane bone in the skull of a single cat, and repetition of his experiments by Melzer and by the Guggenheims [3] has shown that the rather far reaching conclusions drawn from the original work are subject to question.

Holmgren [4] has produced fistulas in the horizontal semicircular canals of monkeys, but he reported that all of them closed except one, in which radium was used. Holmgren has not reported on the early stages of the closure of the fistula in monkeys; so one is unable to visualize from his reports the mechanism of fistula closure. Since he worked with Nager,[5] it was naturally deduced that the experimental fistulas in monkeys were closed by growth of bone from the periosteal layer of the labyrinthine capsule. Nager has repeatedly pointed out, since the work of Ulrich [6] on fractures of the temporal bone was published, that after a fracture through the labyrinthine capsule bony union develops only in the periosteal layer. He suggested, therefore, that if the periosteal bone could be prevented from growing back the endochondral layer would never heal and the fistula would remain patent. Holmgren [4] has experimented with a great number of artificial means for accomplishing this end. He has tried Thiersch grafts, grafts of conjunctiva, fat pads, metal foil, small radium bombs and even laminaria, among others. His success, however, has not been signal with any of these.

Most of the experimental work on fenestration of the labyrinth has been done on human beings. Sourdille,[7] after observing the immediate results of Bárány and Holmgren, has concentrated on the study of various types of skin flaps to keep his fistulas open. He found that mobilization of the skin from the superior wall of the horizontal semicircular canal near its attachment to the drum seemed to work better than

3. Guggenheim, L. K., and Guggenheim, P.: Artificial Fistula in Cases of Otosclerosis, Arch. Otolaryng. **32**:1 (July) 1940.

4. Holmgren, G.: Surgical Therapy for Otosclerosis, in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1938.

5. Nager, F. R.: Demonstration zur Holmgrenschen Otosklerosoperation an Affen, Acta oto-laryng. **27**:350, 1939.

6. Ulrich, K.: Verletzungen des Gehörorgans bei Schädelbasisfrakturen, Helsinki, Mercator, 1938.

7. Sourdille, M., (*a*) cited by Duel, A. B.; Fowler, E. P., and others: Otosclerosis: Résumé of Literature from 1928-1935, Ann. Otol., Rhin. & Laryng. **45**:3 (March) 1936; (*b*) A New Technique in the Surgical Treatment of Severe and Progressive Deafness from Otosclerosis, Laryngoscope **47**:853 (Dec.) 1937.

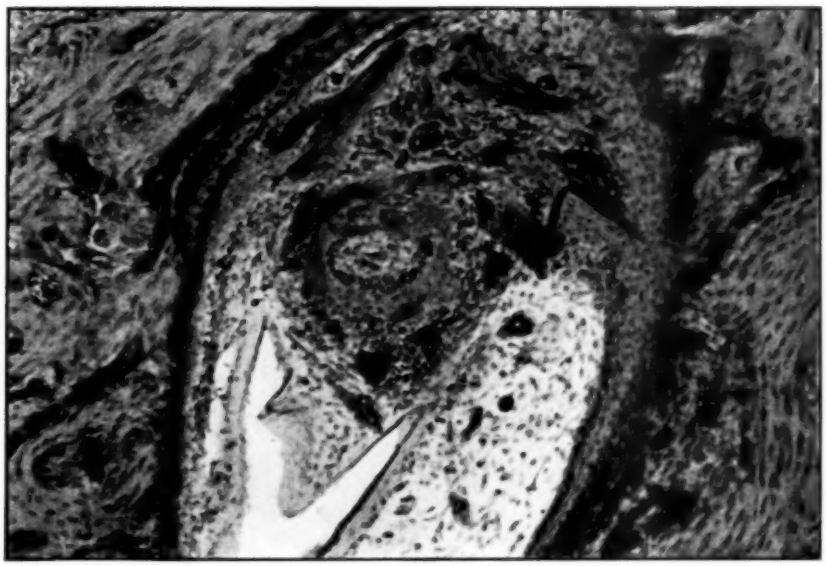

Fig. 1 (animal 517; right ear).—Osteoid tissue and new bone growth in the perilymphatic space of the horizontal semicircular canal beyond the opening of a fistula of thirteen days' duration. Note that the new bone growth takes place from the endosteum and around the bone chips. The structure in the lower left hand corner is the endolymphatic space.

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anything else, if he removed the head of the malleus. That he has not considered the skin of the superior wall alone sufficient for positive purposes is manifested by his continual changes of technic; he has used a flap of scar tissue, a flap treated with solution of formaldehyde or even no flap at all. He has insisted that the incus must be left in place if the best improvement in hearing is to ensue, but he has given no satisfactory explanation of why this is necessary.

Lempert [8] has used a method which is substantially the Sourdille technic reduced to one stage. He has claimed that his endaural incision reduces infection and trauma to tissue and that this is why he is more successful than Sourdille. He has agreed with the statement of Sourdille, however, that the use of the thinnest of skin flaps made from the superior wall of the canal and the drum as well as removal of the head of the malleus and maintenance of the normal position of the incus are essential for good results. He, too, has tried grafts of conjunctiva and Thiersch grafts, but instead of laying them directly on the fistula, as Holmgren did, he has used them in conjunction with a Sourdille flap and has reversed the epithelium of the graft so that it faces toward the lumen of the fistula, while its deeper surface is applied to the connective tissue of the flap (fig. 8).

At complete variance with the theories which activated the attempts just described to keep fenestrae open with Thiersch or conjunctival grafts, placed with their epithelial surface to the lumen of the fistula and nourished by a skin flap, is Lempert's insistence that in the average case it is necessary to cover the fistula with Shrapnell's membrane. This is manifestly an anatomic impossibility, for, even when loose, the membrana flaccida never measures more than 2.2 mm. in height and 4.0 mm. across its base. It averages 1.4 by 3.5 mm. After all, the entire drum averages only 9 mm. in its longest diameter, and the membrana flaccida is generally described as taking up less than one sixth of this total height. These measurements are based on study of 25 unselected vertical sections of the temporal bone. Measurements were made from the short process of the malleus to the roof of the external auditory canal. Since the sectioning is rarely exactly vertical and since any variation from the vertical would increase the measurement for the height of Shrapnell's membrane, it can be seen that, if anything, the foregoing figures tend to exaggerate the height of Shrapnell's membrane rather than to minimize it. The distance from the drum to the prominence of the horizontal semicircular canal is 3 to 3.5 mm. It might be possible

8. Lempert, J.: (*a*) Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **28**:42 (July) 1928; (*b*) Endaural Fenestration of External Semicircular Canal for Restoration of Hearing in Cases of Otosclerosis: Summary Report of One Hundred and Twenty Cases, ibid. **31**:711 (May) 1940.

to stretch the edge of Shrapnell's membrane to the anterior border of a fistula made close to the ampulla of the horizontal semicircular canal if the drum were thoroughly separated from the annulus for a considerable distance and if the head of the malleus as well as the entire incus were removed. I believe, however, that the foregoing figures demonstrate incontrovertibly that with the incus in place Shrapnell's membrane in the human being is always too short to go over the top of the incus, span the intervening space and cover a fistula in the horizontal semicircular canal.

That applying a flap of the extremely thin skin of the superior wall of the canal is not necessary for the maintenance of an open fenestra was demonstrated by a case reported by Kopetzky,[9] in which no skin at all was placed over the operative fistula. Holmgren also has reported cases of this type. Furthermore, I have 2 patients who were operated on, 1 four months ago and the other over a year ago, and in both cases the fistula of the horizontal semicircular canal has remained open though covered simply by the skin of an old, dry radical mastoidectomy cavity. (Incidentally, there was no improvement of hearing in these 2 cases.)

In an attempt to study what happens when an opening is made in the horizontal semicircular canal and covered with skin, 7 large monkeys were operated on and their fistulas examined at various stages of the healing process. Summaries of the protocols for these monkeys are given in the next section of this paper; in each instance the number of days from operation to autopsy is indicated.

EXPERIMENTAL OBSERVATIONS

PROTOCOL 1.—*Animal 521, a baboon weighing 8 Kg.; operation on left ear, with animal under anesthesia induced by 30 mg. of pentobarbital sodium per kilogram; death during operation.*

A semilunar postauricular incision was used. The skin and the periosteum were elevated well forward into the zygoma. The posterior portion of the cartilaginous external auditory meatus was severed near its attachment to the bone. The mastoid was entered and exenterated with curets. The posterior and superior meatal walls were skeletonized and separated from the skin of the canal with periosteal elevators. A small periosteal elevator was slipped through the fissure of Rivinus, and the drum was separated from the annulus membranae tympani but left attached to the meatal skin. The bone of the canal wall was removed with small biting forceps. At this point a large vein was injured in an attempt to obtain space in the zygomatic region. There was profuse bleeding, and suddenly the monkey died. The operation was continued, however, and a fistula made in the labyrinth with a dental polishing burr. The endosteum was severed with a cataract knife toward the solid angle but left intact on the

9. Kopetzky, S. J.: Labyrinthine Fenestration to Improve Hearing, Laryngoscope **49**:1064 (Nov.) 1939.

peripheral side of the fistula. A strip of conjunctiva was placed over the fistula and an attempt made to hold it in place with the flap from the posterior wall of the canal, but it was so slimy and slippery that after the flap was turned over I was not at all certain that it actually covered the fistula. Flap and graft were held in place with paraffin treated with petrolatum. Autopsy was performed at once. No cause for the sudden death was apparent. The temporal bones were placed in a 10 per cent concentration of solution of formaldehyde U. S. P.

Microscopic Examination.—The fistula was almost completely filled with loose, folded subconjunctival tissue. The conjunctival graft had become reversed, that is, its epithelium did not face the lumen of the canal. The endolymphatic system was intact but lay deep in the canal as if it had been pushed in by the conjunctival graft or had retracted with the opening of the canal or the incision of the endosteum. The flap of skin and subcutaneous tissue from the wall of the membranous canal, which covered the conjunctival graft, was extremely thin; it averaged about 0.5 mm. in thickness but in some places was thinner. (The thickness of this flap in monkeys reported later was 1.5 to 2.0 mm. The difference was undoubtedly due to postoperative edema.) There were a moderate number of bone chips under the flap. The edges of the opening in the labyrinth were smooth, but did not look strikingly smoother than edges made with a chisel.[4]

PROTOCOL 2.—*Animal 518, a giant rhesus monkey weighing 8.5 Kg.; right ear operated on, with animal under anesthesia induced by 30 mg. of pentobarbital sodium per kilogram; autopsy three days after operation.*

The usual postauricular incision was used, and the cartilaginous meatal wall was severed for exposure. The cavity was exenterated with bone burrs, gages and curets. The skin covering the bony meatal wall was elevated with small periosteal elevators and separated from the annulus at its attachment to the drum without difficulty, but when the bony wall was taken down part of the flap was caught and torn slightly near its anterior edge. The ossicles were left in place. A fistula was made in the promontory of the horizontal semicircular canal with a dental polishing burr, and the edges were smoothed and widened with a Spratt curet until the opening was 2.5 by 1 mm. The torn skin flap was mobilized by separating it above and below with sharp scissors. It was then placed over the fistula in the canal and held there with two small balls of petrolatum gauze.

Microscopic Examination.—The fistula was not closed with bone, but there was acute labyrinthitis in and near the operative opening. Polymorphonuclear neutrophils could be seen between the epithelial cells of the endolymphatic membranous labyrinth. There was much inflammation about scattered bone chips in and around the lumen of the canal.

PROTOCOL 3.—*Same animal as in protocol 1; operation on right ear, with animal under anesthesia induced by 30 mg. of pentobarbital sodium per kilogram; autopsy twelve days after operation.*

A semilunar postauricular incision was used. The skin and the periosteum were elevated backward and forward to expose the mastoid in the zygomatic region. The posterior portion of the cartilaginous external meatal wall was severed near its attachment to the bone. The mastoid cavity was exenterated with curets and surgical burrs. There was much bleeding from a large vein in the uncovered dura of the middle fossa. The bony wall of the external auditory canal was taken down with bone forceps, and a spade-shaped flap was fashioned

from the skin which covered its superior and posterior portions. This skin flap was separated from the annulus with the tiny bent periosteal elevator designed by Sourdille. The head of the malleus was separated from the incus and cut away at the neck with cuticle scissors. Under a dissecting microscope, a fistula 4 by 1 mm. was made with a dental polishing burr at the upper, outer edge of the horizontal semicircular canal. A transparent, gray tube thought to be the membranous labyrinth was apparent at first, but when the bone dust had been cleaned out with cotton applicators it suddenly disappeared and even with 7 × magnification was never again definitely visualized. There was little evidence of bone dust when the previously prepared flap was placed over the fistula in

Fig. 2 (animal 517; left ear).—Fistula of seventeen days' duration. Note that the opening is filled with small cysts and a hyperplastic finger of epithelial tissue from the skin flap.

the horizontal semicircular canal and fixed there with paraffin treated with petrolatum. The skin was closed with black silk mattress sutures, with a small amount of silk showing.

Microscopic Examination.—The fistula was not closed with bone. It was covered by thick, swollen mucoperiosteum containing a few bone chips. There was relatively little reaction about the bone chips.

PROTOCOL 4.—*Animal 517, a giant rhesus monkey weighing 8 Kg.; operation on right ear, with animal under anesthesia induced by pentobarbital sodium; autopsy thirteen days after operation.*

The operative procedure followed was identical with that previously described except that the fistula was only 2.5 mm. in length; the flap was not torn on removal of the head of the malleus.

Microscopic Examination.—The fistula was open but was filling up with osteoid tissue from bone chips and endosteum. There was not so much growth from the edge of the periosteal capsule as is usual at the operative site. Elsewhere in the mastoid cavity there was considerable new growth of bone. Within the lumen of the canal there was some inflammation of the perilymphatic and endolymphatic systems at the operative site, but elsewhere these appeared normal (fig. 1).

PROTOCOL 5.—*Same animal as in protocol 2; operation on left ear; autopsy fifteen days after operation.*

Fig. 3 (animal 522; left ear).—Fistula of twenty-two days' duration filled with connective tissue and a large chip of old bone. Cystic material in the skin flap in the upper right hand corner is due to rolling of the edges of the skin.

The technic of operation was the same as previously described; the head of the malleus was removed. The flap was short and barely covered the fistula, which in this case was made entirely with Spratt curets and a small mastoid gage and measured 3 by 1 mm. The flap was held in place with paraffin gauze. Three days after operation the monkey had a foul-smelling cavity, acute auricular perichondritis and paralysis of the left side of the face. When, at that time, the paraffin gauze was removed from the mastoid cavity, the meatal skin flap was found to be free from the fistula and partially necrotic. The necrotic portion of the flap was cut away.

Microscopic Examination.—There was low grade acute labyrinthitis in the region of the operative wound. The endolymphatic system seemed intact in all

but the few sections through the operative area. There were many bone chips in the canal, about which there was moderate osteoid regeneration. There was no bony closure of the fistula.

PROTOCOL 6.—*Same animal as in protocol 4; operation on left ear; autopsy seventeen days after operation.*

The usual operative procedure was followed, with removal of the head of the malleus. The flap was slightly torn in trying to stretch it over the fistula, and three days after operation part of it came away with the petrolatum gauze used to hold it in place.

Microscopic Examination.—The fistula was open, but there was a small amount of osteoid regeneration about from bone chips as well as from the periosteal and the endosteal capsule. The new growth of bone was much less than usual, for most of the fistulous opening was taken up by a series of cysts containing degenerating epithelium (fig. 2). These originated in the infected skin flap.

PROTOCOL 7.—*Animal 522, a Macacus rhesus monkey weighing 2 Kg.; left ear operated on, with animal under anesthesia induced by pentobarbital sodium; autopsy twenty-three days after operation.*

The usual postauricular incision was used and exenteration of the mastoid was carried out. The horizontal semicircular canal was opened with a polishing burr, and a fenestra 3 by 1 mm. was formed. The endosteal lining was not cut, but it seemed to be slightly stripped from the bony wall of the canal. The fistula was cleared of bone dust, except at its anterior end. The flap was slightly torn on removal of the head of the malleus. The flap was held in place with paraffin.

Microscopic Examination.—The fistula was filled with connective tissue. There was no growth of bone. The endolymphatic system was destroyed in the region where the fistula was made, but it looked normal elsewhere except for slight swelling of its epithelial lining. Bone chips were present in the perilymphatic space. The curled skin flap was degenerating into a cystic mass (fig. 3).

PROTOCOL 8.—*Animal 523, a Mona monkey weighing 3.1 Kg.; operation on left ear, with animal under anesthesia induced by 20 mg. of pentobarbital sodium per kilogram supplemented by 0.2 Gm. of morphine sulfate; autopsy thirty-five days after operation.*

The usual semilunar incision was employed, and exposure of the horizontal semicircular canal and creation of a meatal flap were effected. The mastoid was even more pneumatized than those of rhesus monkeys and the incus less hidden because of the extent of the zygomatic air cells. The head of the malleus was separated from the incus and cut away with cuticle scissors. The fistula measured approximately 2.5 by 1 mm. and was made with gage, mallet and dental chisels. A few tiny remnants of wet bone chips remained under the edge of the fistula and could not be removed. The flap from the posterior wall was placed over the fistula and allowed to lie in place, held only by a small amount of blood coagulum. Nothing was placed in the cavity; the skin and the subcutaneous tissue were closed in the usual way with mattress sutures and black silk.

Microscopic Examination.—The fistula, in spite of the fact that it extended halfway to the inner edge of the horizontal canal, was largely closed by actively growing, short fiber web bone. Some of this seemed to spring from the endosteum, but most of it definitely originated in the connective tissue covering the fistula. This connective tissue was the subepithelial lining of the middle ear cavity and was highly inflamed. It was filled with leukocytes and fibroblasts and contained

many engorged capillaries. The epithelium had become high columnar in type, even in the region directly covering the fistula, while it is normally quite flat. In the section in which complete bony closure had not taken place there was a pocket of this epithelium within 0.5 mm. of the endolymphatic membranous labyrinth, which was intact. A few bone chips about which active new bone growth was taking place were present in the connective tissue covering the fistula.

PROTOCOL *9.—Animal 524, a Macacus rhesus monkey weighing 2.5 Kg.; left ear operated on, with animal under anesthesia induced by 30 mg. of pentobarbital sodium per kilogram supplemented by 0.2 Gm. of morphine sulfate and a few whiffs of ether twice during operation; autopsy thirty-nine days after operation.*

Fig. 4 (animal 524; left ear).—Closure of fistula and invasion of the perilymphatic space with new bone from the endosteum; thirty-nine days after operation.

The usual method was followed to expose the horizontal semicircular canal, remove the head of the malleus and mobilize the flap. A fistula, 1 by 2.5 mm., was made with a dental polishing burr. It was believed that the endolymphatic system was injured during operation, for it disappeared and was never again visualized. The flap was torn in trying to pull it over the fistula; so it was not put in place at all.

Microscopic Examination.—The entire perilymphatic space in the region of the fistula was filled with bone, which was beginning to form into lamellar systems. The endolymphatic system seemed to be intact (fig. 4).

PROTOCOL 10.—*Same animal as in protocol 9; right ear operated on; autopsy fifty-one days after operation.*

The usual method of exposure was followed. A fistula, 1 by 2.5 mm., was made with a dental burr, and the endosteum was cut on the solid angle side under a dissecting microscope. There was some question whether the endolymphatic system was intact after this. The bone dust was cleared away as well as possible, but a small amount worked its way deep into the canal and could not be removed with cotton swabs. A small piece of skin from an upper eyelid was placed over the fistula, with the epithelial side down and was held in place by the skin

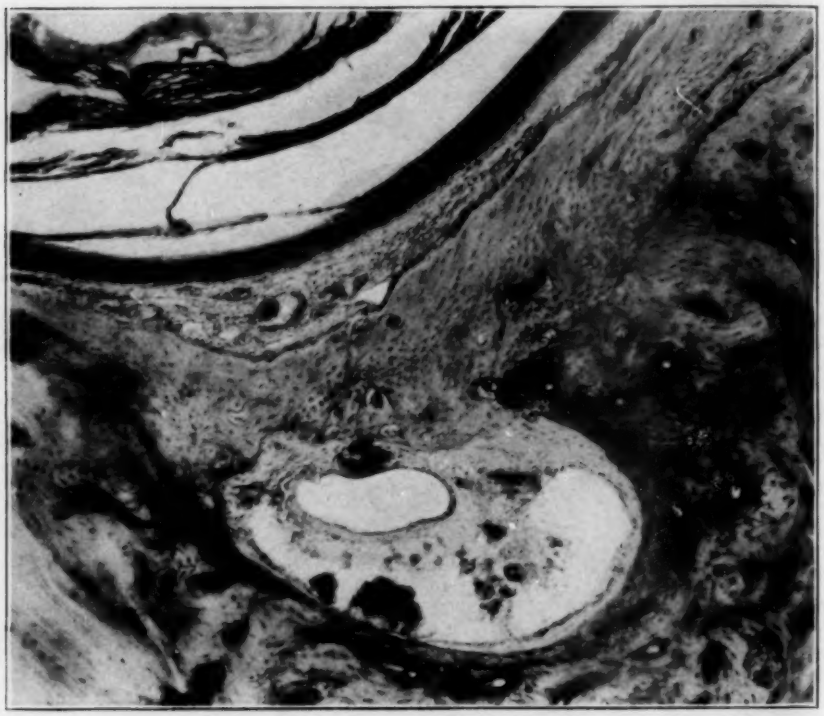

Fig. 5 (animal 524; right ear).—Complete closure of a fistula with new bone; fifty-one days after operation.

flap from the posterior and superior portions of the external auditory canal. The latter flap was held in place by a wad of paraffin treated with petrolatum.

Microscopic Examination.—The fistula was completely closed with periosteal bone, bone chips and endosteal growth. Infection in the region of the fistula was not evident, although there was a small amount elsewhere in the cavity. The skin graft from the upper eyelid was apparently absorbed (fig. 5).

PROTOCOL 11.—*Same animal as in protocol 7; the right ear was operated on; autopsy fifty-four days after operation.*

The usual operative procedure was followed except that the fistula was made on the convexity of the canal, with curets only. The flap was held in place with paraffin.

Microscopic Examination.—The fistula was closed with dense bone. There was considerable infection throughout the mastoid cavity. No epithelium was evident in the region of the fistula. Apparently the skin flap had been completely autolyzed (fig. 6).

PROTOCOL 12.—*Same animal as in protocol 8; right ear operated on, with animal under anesthesia induced by 20 mg. of pentobarbital sodium per kilogram supplemented by 0.2 Gm. of morphine sulfate; autopsy fifty-six days after operation.*

The usual semilunar incision was used to expose the horizontal semicircular canal. The mastoid was more pneumatized than those of rhesus monkeys and

Fig. 6 (animal 522; right ear).—Complete closure of labyrinthine fistula with new bone; fifty-four days after operation. Note the bone chips, the infected connective tissue over the fistula and the absence of epithelial covering. The structure in the upper left hand corner is curled stratified squamous epithelium from an unsuccessful flap.

the incus less hidden. The incus and the malleus were not disturbed. Part of the exenteration of the mastoid was done with a burr. A fistula, 1 by 4 mm., was made with a polishing burr. The bone dust and chips were only partially cleaned out. The endosteum was cut on a curve toward the solid angle. The bony edges were polished with a gold polishing burr. A small piece of conjunctiva was placed over the fistula and secured by a skin flap from the posterior wall of the canal, which was held in place with paraffin. The wax was left in

place for four weeks, after which time the cavity appeared perfectly dry and smoothly epithelized.

Microscopic Examination.—The perilymphatic cavity was filled with bone, but the endolymph was still intact. Such a course as that in this case would account for a positive reaction to the fistula test for vertigo without improvement in hearing (fig. 7).

PROTOCOL 13.—*Animal 520, a female rhesus monkey; left ear operated on, with animal under anesthesia induced by pentobarbital sodium; autopsy seventy-two days after operation.*

The usual postauricular incision was made, and the wall of the membranous meatal canal was exposed with curets and forceps. The head of the malleus

Fig. 7 (animal 523; right ear).—Perilymphatic space filled with new bone. The endolymphatic space is open and covered only by a pad of infected soft tissue. In the presence of conduction deafness, this type of result might cause a positive reaction to the fistula test for vertigo but no improvement in hearing following fenestration of the labyrinth.

was removed. A fistula, 1 by 3 mm., was made at the top of the horizontal semicircular canal with a polishing burr. A satisfactory untorn flap was secured. A Thiersch graft from an upper eyelid was placed under the flap, and the whole was held in place with paraffin. The paraffin was removed forty-two days after operation, at which time it exuded slightly through the wound in the posterior wall of the canal. The cavity looked healed and dry, as did the flap.

Microscopic Examination.—The fistula was still open. There were pockets lined by cuboidal epithelium from the middle ear close to the open fistula. There was some osteoid regeneration along the edges of the fistula, which suggested that it might eventually close (figs. 8 and 9). There was complete absorption of the Thiersch graft in the region of the fistula, but there were two unabsorbed areas in which the skin was beginning to form into typical epidermal inclusion cysts (cholesteatomas) (fig. 10).

PROTOCOL 14.—*Same animal as in protocol 13; right ear operated on; autopsy eighty-five days after operation.*

Fig. 8 (animal 520; left ear).—Fistula (*F*) not yet filled in with bone seventy-two days after operation; low magnification. Note that the fistula is covered with connective tissue and low cuboidal epithelium from an outpouching middle ear pocket (*E*). Note the junction of the squamous epithelium of the flap and the cuboidal epithelium from the mastoid (*J*). Note the remnants of a Thiersch graft developing into epidermoid inclusion cysts (*C*). Compare with figures 9 and 10.

The usual postauricular incision was made and the membranous canal exposed. There was little bleeding. The membranous canal was prepared with puncture of the flap above and below. The ossicles were exposed, and the incus was dislocated during the attempt to cut off the head of the malleus; so the incus was removed with the head of the malleus. A fistula, 1 by 2.5 mm., was made on the periphery of the horizontal semicircular canal with a polishing burr;

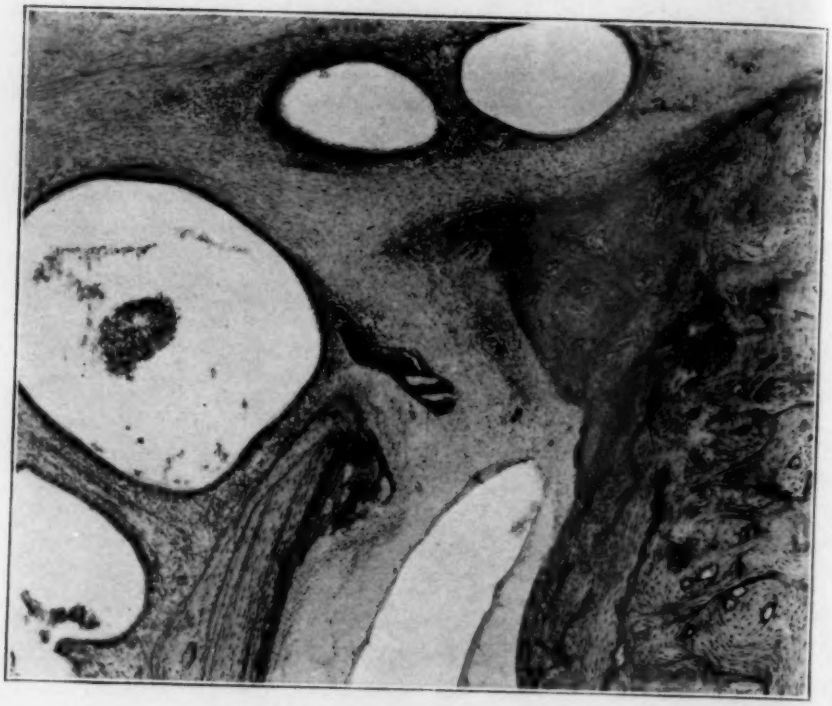

Figure 9

Figure 10

(See legend of figures 9 and 10 on opposite page)

there was apparent destruction of the membranous labyrinth, and some bone dust could not be extricated from the lumen of the canal. A satisfactory, although narrow, flap was placed over the canal. No packing was applied.

Microscopic Examination.—Sections were cut parallel to the opening of the fistula, so that it was difficult to visualize. Apparently it was completely closed, for the perilymphatic space in the region of the fistula was entirely filled with new bone. The endolymphatic system, however, was intact.

COMMENT

These protocols show that closure of labyrinthine fistulas in monkeys is almost the rule, whether they are made with a polishing burr or with a curet, and yet bony closure of fistulas made with either instrument may not occur. The periosteal bone of the monkey seems to grow even more rapidly and more constantly than that of human beings. From the work of Nager [10] and Ulrich,[6] who showed that fractures of the labyrinthine capsule do not heal in such bone, one would expect that the only healing of labyrinthine fistulas would be from the periosteal capsule. One sees, however, that although the most marked growth is from the periosteal capsule and its periosteum, definite growth occurs from the endosteum and probably from the edge of the endosteal capsule (figs. 4 and 5). Growth from the endochondral layer has also been observed (figs. 1 and 7).

There is also always some osteoid tissue about any bone chips which are present in the connective tissue covering the fistulas (figs. 1 and 6). This observation is in harmony with those of Murray [11] on the healing of fractures.

The presence of pockets of low cuboidal epithelium covering the open fistula in the left ear of animal 520 (fig. 8) and the epithelial cysts which seemed to keep the fistula in the left ear of animal 517 open suggest that the presence of epithelium reduces regeneration of bone

10. Nager, F. R., in Fowler, E. P., Jr.: Medicine of the Ear, New York, Thomas Nelson & Sons, 1939.

11. Murray, C. R.: The Repair of Fractures, Minnesota Med. **13**:137 (March) 1937.

EXPLANATION OF FIGURES 9 AND 10

Fig. 9 (animal 520; left ear).—High magnification of fistula shown in figure 8, showing proximity of the epithelium of the middle ear and large blood vessel in the enclosed portion of the fistula. There is considerable new bone growth, but it is possible that this fistula may remain permanently open.

Fig. 10 (animal 520; left ear).—Cyst formation (cholesteatoma from unresorbed portion of a Thiersch graft from the eyelid placed under a flap from the posterior auditory meatal wall); high magnification. Compare with figure 8 (*C*).

(fig. 2). When stratified squamous epithelium from a Thiersch graft was placed under the usual flap in the posterior meatal wall, it was absorbed in the region of the fistula but often balled up into epidermal cysts elsewhere. From this one could deduce that the use of Thiersch grafts is fraught with the danger of producing unwanted epidermal inclusion cysts (cholesteatomas).

The covering of the open fenestra in the left ear of animal 520 by epithelium from the middle ear (fig. 8) suggests why hearing is so much better in human beings when the incus is left in place. (This has never before been satisfactorily explained.) I believe that when the incus is not dislocated it holds the skin flap sufficiently away from the fistula so that cuboidal epithelium on the back of the flap can grow backward and medially to meet the epithelium from the front of the horizontal semicircular canal as it grows backward and laterally. Thus a sort of pouch is formed between the body of the incus and the fistula, and the covering of the fenestra becomes mucoperiosteum from the middle ear rather than the comparatively thick skin and subcutaneous tissue of the superior wall of the canal. A thin covering of low cuboidal epithelium over a fistula in the perilymphatic labyrinth would be much more elastic and presumably would function better in improving the hearing than a fistula covered by any other type of tissue. Incidentally, a Thiersch graft under the flap tends to permit the fast-growing cuboidal epithelium of the middle ear to cover the fistula and will hold the flap out of the way until this has occurred. This explanation might account for the success of the procedure as reported for human beings, but unless it is certain that all of the Thiersch grafts will later be absorbed it is hardly to be recommended since it is known that those portions which are not absorbed will develop into epidermal inclusion cysts, so-called cholesteatomas (fig. 8).

The monkeys' mastoid cavities in which infection developed, and especially those in which the infection entered the perilymphatic space, showed much faster regeneration of bone than the cavities which were relatively sterile. The localized labyrinthitis present in all of the monkeys near the operative site undoubtedly explains the vertigo of patients who have been operated on and also why their reaction to the fistula test for vertigo tends to become less positive a few days after operation. When the localized labyrinthitis subsides, the reaction to the fistula test again becomes positive unless bony closure has begun to take place. In the case of human beings infection should obviously be carefully guarded against, both during the operation and during subsequent dressings.

The questions brought up by the data given in this paper and many others which may arise in regard to this complicated procedure should be investigated by further animal experimentation.

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SPHENOID SINUS

ANATOMIC STUDY, WITH CONSIDERATION OF THE CLINICAL SIGNIFICANCE OF THE STRUCTURAL CHARACTERISTICS OF THE SPHENOID SINUS

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CHICAGO

The sphenoid sinus reflects its anatomic relations in its walls. Adjacent structures, present before the development of the sinus, produce irregularities in the walls of the sinus as the cavity grows to contact and pass beyond them. Often the extension is such as to form recesses on either side of an elevation, and in the well pneumatized cavities the walls commonly present a continuous sequence of depressions and elevations. In such cases only a thin bony plate separates the sinus from the adjacent structures, and the intimate relationship thus established contributes much to the clinical importance of the sphenoid sinus.

The structures usually involved in this relationship are the blood vessels and nerves which course alongside the sinus on their way to or from the cranial cavity.

Also closely related to a sphenoid sinus are its fellow sinus, contiguous posterior ethmoid cells and the sella turcica, which protrudes downward into the sinus.

The sinus is not limited to the body of the sphenoid bone. Extension into adjacent bony processes occurs regularly. Recesses are commonly found in the greater wing and the base of the pterygoid process. Others extending anteriorly from the sphenoid cavity are often sufficiently large to occupy a considerable portion of the ethmoid field. Occasionally there are extensions into the lesser wing of the sphenoid bone and into the anterior and posterior clinoid processes; in some instances a posterior extension reaches the basilar portion of the occipital bone.

The sphenoid sinus is not always a large thin-walled cavity with extramural extensions. In many cases the body of the sphenoid bone is only partially pneumatized, and the cavity is small with thick walls. In other cases retardation of the growth of one sinus permits encroachment by the other sphenoid cavity or by a posterior ethmoid cell. In certain instances the sphenoid area of one side may be almost completely taken over by the companion sinus or by a large ethmoid cell, which itself may expand into adjacent bony processes.

The various types of encroachment, the irregularities in the sinus walls and the pneumatization of adjacent bony processes occur with such frequency that all may be regarded as typical of the anatomy of the sphenoid sinus. A clear understanding of these various characteristics and others of the normal anatomy of the sinus may be reached only by examination of large series of specimens from cadavers.

Such an examination was recently undertaken at the University of Illinois College of Medicine. A study was made of 100 adult specimens taken at random from the anatomy laboratory and from the collection in the department of otolaryngology. A study was also made of serial sections from fetal heads of various ages, and a few specimens from the newborn were examined grossly. In this investigation an effort was made to study: (1) the origin and development of the sphenoid sinus; (2) the partial septums and their developmental and clinical significance; (3) the dimensions of the adult sinus, with a view to establishing the average; (4) the anatomic relations of the sinus both with adjacent nerves and blood vessels and with neighboring nasal sinuses; (5) the frequency of recesses, both intramural and extramural; (6) the sphenoid ostium as to its location in the sphenoethmoid recess, its dimensions and its clinical accessibility; (7) the clinical significance of the anatomic findings.

ORIGIN AND DEVELOPMENT OF THE SPHENOID SINUS

Early in the third fetal month the primitive sphenoid sinus is apparent as an invagination of the nasal mucosa into the posterior portion of the cartilaginous nasal capsule (fig. 1). Expansion downward and backward soon forms a pouchlike cavity in the cartilage, the wall of which is called the sphenoid turbinate (ossiculum Bertini). Ossification of this structure begins during the fifth month and is fairly well advanced at the time of birth (fig. 2). It is not, however, until the third or fourth year that with the resorption of intervening cartilage the bony turbinate becomes attached to the body of the sphenoid bone and the cavity becomes definitely sphenoid.[1]

Early pneumatization of the sphenoid bone occurs in an inferior posterolateral direction. Soon a thinning of the lateral wall takes place, causing contact with certain cranial nerves and blood vessels. The sinus floor becomes hollowed out to reach the vidian (pterygoid) canal in the sixth or seventh year, and two or three years later medial expansion by both sphenoid sinuses causes a thinning out of the intersinal septum (fig. 3).[2]

1. Onodi, A.: The Accessory Sinuses of the Nose in Children, translated by C. Praunitz, London, John Bale, Sons & Danielsson, Ltd., 1911.

2. Davis, W. B.: Nasal Accessory Sinus in Man, Philadelphia, W. B. Saunders Company, 1914.

The growth of the sinus into the body of the sphenoid bone is best understood when there is familiarity with the developmental stages of the bone itself. The sphenoid bone is a complicated structure with various

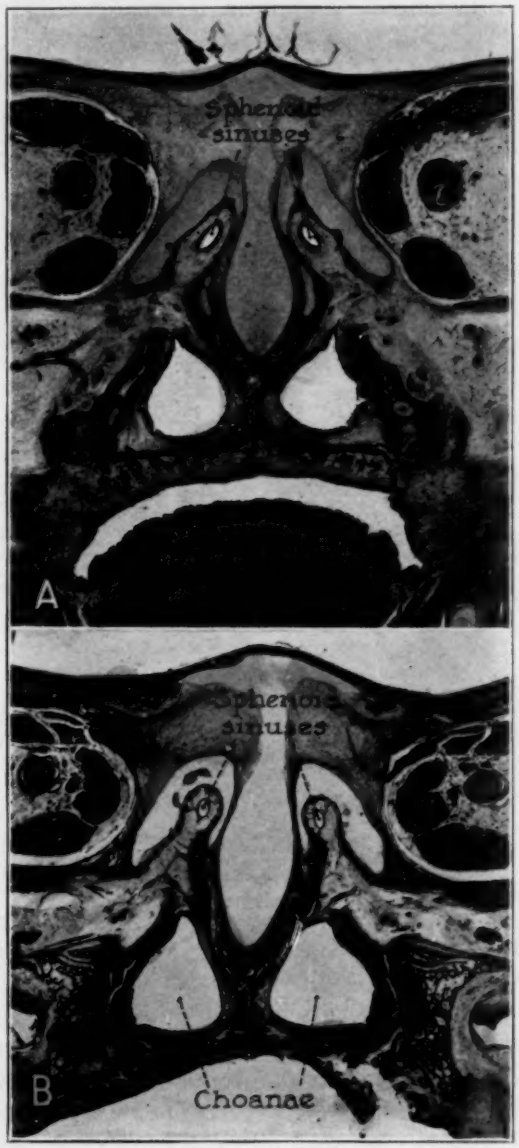

Fig. 1.—Photomicrographs of a coronal section through the nose of a fetus; *A*, aged 17½ weeks; *B*, aged 6¼ months.

outlying processes, each process developing from its own ossification center. These centers in most instances arise within cartilage (endo-

chondral). They appear early in fetal life, and union of the various component parts of the bone begins before birth. At that time, the lesser wings (orbitosphenoid) unite with that portion of the body (presphenoid) which lies between them, the greater wings (alisphenoid) unite with the basisphenoid, and the presphenoid unites with the basisphenoid (fig. 4). At the various points of union fusion planes develop, which offer resistance to the pneumatizing process of the growing sinus.

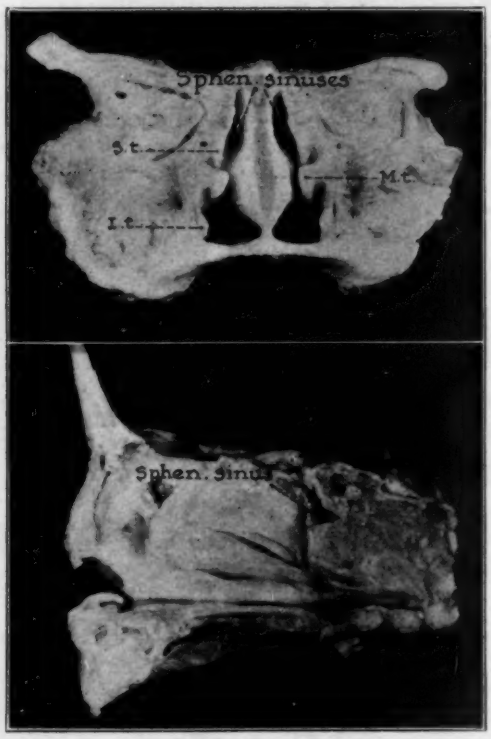

Fig. 2.—Sphenoid sinus of the newborn. These views show the rudimentary sinuses as pouchlike cavities in the terminal nasal cartilage (ossiculum Bertini).

The deflection of the intersinal septum from the midline and consequent overlapping of one sphenoid sinus by the other may be accounted for by a retardation of the growth of the smaller sinus at the prebasisphenoid synchondrosis (Van Alyea,[3] fig. 9). Cope[4] expressed this view in

3. Van Alyea, O.: The Ostium Maxillare, Arch. Otolaryng. **24**:553 (Nov.) 1936.

4. Cope, V. Z.: Internal Structure of the Sphenoid Sinus, J. Anat. & Physiol. **51**:127, 1917.

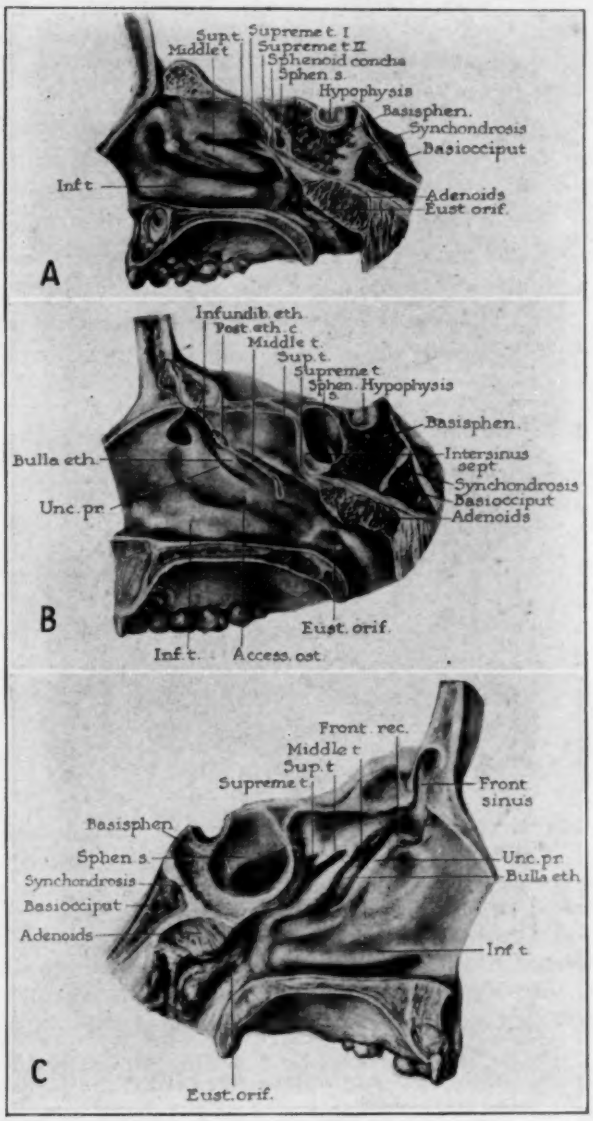

Fig. 3.—Dissections by Davis [2] showing various stages in the development of the sphenoid sinus: *A*, lateral view of the sinus of a child aged 1 year 10 months 13 days. At this age the sinus is a pouchlike cavity in the cartilaginous sphenoid concha. *B*, lateral view of the sinus of a child aged 5 years 10 months 22 days. Invasion of the bone by the sinus has taken place. *C*, lateral view of the sinus of a child aged 10 years 7 months. *B* and *C* both show considerable lateral expansion with beginning formation of partial septums. In *C* expansion has reached the hypophysis and posteriorly is approaching the spheno-occipital synchondrosis.

1917 and maintained that even though the synchondrosis has disappeared, the bone at the fusion line is denser and more resistant than that on either side of the line. Congdon [5] agreed that there is a noticeable tendency for intersinal septums to take the course of certain fusion planes. He applied the same theory to the small anteriorly placed sinuses, which he thought were halted early in development at the concha-presphenoid junction. He classified the sphenoid cavities into four types based on the arrangement of the ossification centers in an anteroposterior series in either half of the bone. The types are: (1) conchal sinuses, those anterior to the concha-presphenoid plane (fig. 12 *D*); (2) presphenoid sinuses, those extending into the presphenoid but not into the basisphenoid (Van Alyea,[3] fig. 9); (3) basisphenoid sinuses, those extending into but not beyond the basisphenoid (figs. 5, 11 and 13); (4) occipitosphenoid

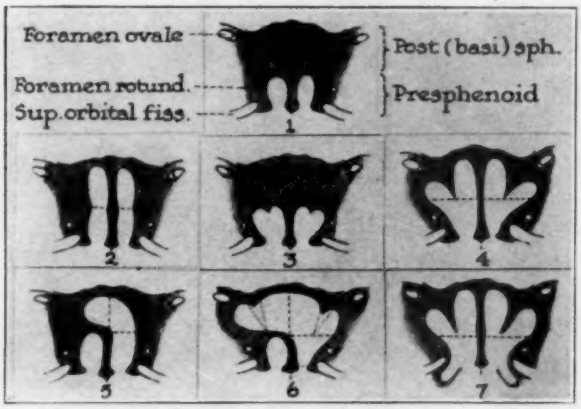

Fig. 4.—Diagram of the ground plan of the different types of sinuses (modified from Cope [4]). The cavities are shown as seen from above. *1*, presphenoid type. *2*, post (basi) sphenoid type. *3*, presphenoid type with small lateral recesses. *4*, type with marked lateral pterygoid recesses and partial septums. *5*, right presphenoid type; the left sinus occupies the whole of the postsphenoid. *6*, the right sinus is presphenoid; the left occupies the entire postsphenoid with lateral (pterygoid) recesses bilaterally. *7*, type showing lateral (pterygoid) and anterior sphenopalatine recesses.

sinuses, those extending into the basilar part of the occipital bone (fig. 8). In Congdon's study of 181 specimens, 9 (5 per cent) were classified as conchal, 8 (4.5 per cent) were undetermined as between the conchal and presphenoid types, 43 (23.5 per cent) were presphenoid, and the remaining 121 (67 per cent) were basisphenoid and occipitosphenoid, with a preponderance of the basisphenoid type.

5. Congdon, E. D.: The Distribution and Mode of Origin of Septa and Walls of the Sphenoid Sinus, Anat. Rec. **18**:97-123, (March) 1920.

Cope's observations were similar to Congdon's, although he recognized only three types. Of his 289 specimens, 72 (25 per cent) ended in front of the basisphenoid, 155 (54 per cent) had an extensive posterior expansion, and 62 (21 per cent) occupied the intermediate zone. These two reports indicate that there is a tendency on the part of the sphenoid to extend posteriorly in the majority of cases and that there would be many more in that group if progress backward were not halted by the compact bone of the various planes of fusion. A normal growing sinus should reach the basisphenoid before the tenth year and attain its mature size at the fourteenth year.

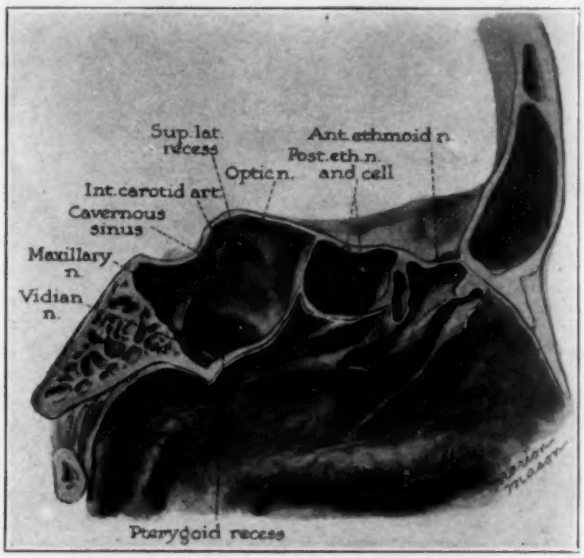

Fig. 5.—Irregularities of the sphenoid walls. The characteristic triangular and oval appearance of the lateral recesses is well illustrated in this specimen.

ANATOMIC OBSERVATIONS

In the present investigation the examination was of one side only in 78 heads and bilateral in 11. The heads examined had all been split in the midsagittal plane, and the examination of the interior of the sinuses was made in most instances after removal of the intersinal septum. In a few cases the septum was left intact, and openings were made in the roof or the lateral wall.

SIZE OF SINUS

The sphenoid sinus is most variable in its anteroposterior dimension. In the present investigation the length of the sinus varied from 4 to

44 mm., with an average of 23.2 mm. for the 100 specimens. Transversely, the main barrier to growth is the intersinal septum. This, although in the midline anteriorly, commonly deviates to one side or the other posteriorly, thereby causing a considerable increase in the width of one sinus over the other. In the 100 specimens the range in width

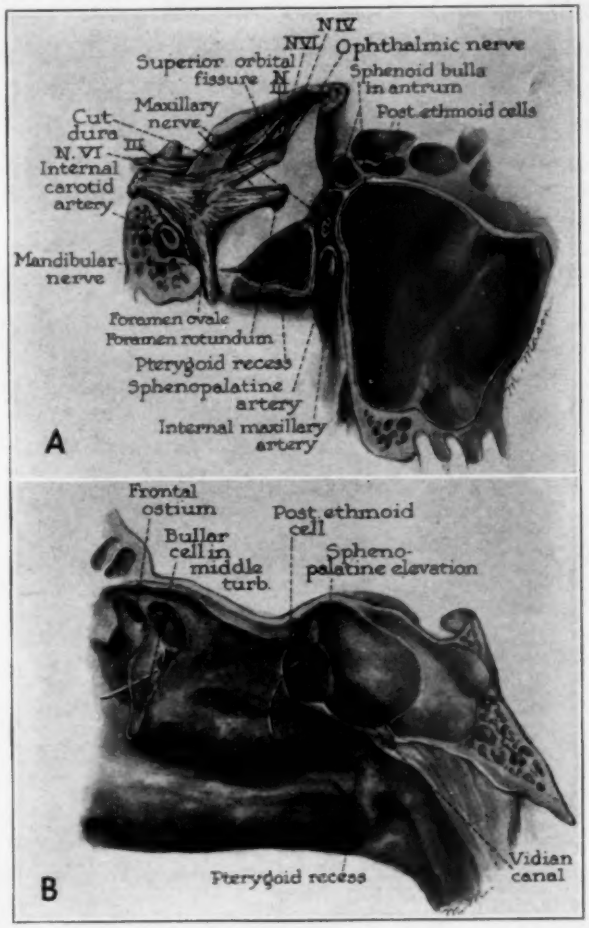

Fig. 6.—*A*, Sphenoid bulla in antrum. In the specimen illustrated the sphenoid sinus has expanded anteriorly over the sphenopalatine foramen and laterally to form a bulge in the medial wall of the maxillary sinus. *B*, view from the nasal side.

was from 2.5 to 34 mm., with an average of 17.4 mm. In height the average was 19.5 mm., the extreme dimensions being 5 and 33 mm.

The findings reported herewith vary but slightly from those of Loeb,[6] Schaeffer[7] and Barnhill[8] and are indicative of what might be expected in an examination of any large series of specimens.

ADJACENT NERVES AND BLOOD VESSELS

In some specimens the sphenoid walls present an even surface, unaffected by adjacent anatomic structures. This applies not always to the small thick-walled cavities but to an occasional large one as well (fig. 11). On the other hand, irregularities in the walls of some sinuses are so numerous as to display an intimate relationship between the sinus and all of the nerves and blood vessels in the sphenoid area (fig. 5).

Those structures which contribute most prominently to the relief in the walls of the sphenoid sinus are the internal carotid artery and the

TABLE 1.—*Elevations in the Sphenoid Sinus Wall*

Nerve or Blood Vessel Causing Elevation	Pronounced	Slight	In Posterior Ethmoid Cell	In Opposite Sphenoid Sinus
Internal carotid artery (partial)	53*	12	4	4
Internal carotid artery (complete)	14	3	..	..
Optic nerve	40	7	5	1
Vidian nerve	36	12	..	..
Maxillary nerve	40	2	1	..
Nerves in the superior orbital fissure	34	8	1	..
Sphenopalatine ganglion	30	8	2	..
Mandibular nerve	4	9 contacts	..	..

* All numbers indicate per cent.

vidian, the optic and the maxillary nerves. Less pronounced is the bulge caused by the group of structures within the cavernous sinus which course along the lateral sphenoid wall and pass through the superior orbital fissure. In this group are the opthalmic, oculomotor, trochlear and abducent nerves. Not infrequently a mound may be observed in the floor anteriorly, which indicates the location of the sphenopalatine foramen (fig. 6) and of the nerves and vessels which traverse it; in a few instances a slight elevation is produced in the lateral posterior inferior angle by the mandibular nerve (fig. 6).

Table 1 shows the frequency with which the various irregularities occurred in the 100 specimens.

6. Loeb, H. L.: Operative Surgery of the Nose, Throat and Ear, St. Louis, C. V. Mosby Company, 1916, vol. 1.

7. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Naso-Lacrimal Passageways and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

8. Barnhill, J. F.: The Nose, Throat and Ear, New York, D. Appleton and Company, 1928, p. 141.

THE INTERNAL CAROTID ARTERY

The internal carotid artery leads the others not only in frequency but also in degree of projection into the sinus (figs. 5, 7 and 9). A carotid elevation was present in 65 of the 100 specimens; in 53 the condition was pronounced. In 14 of the 53, the vessel could be traced throughout its entire serpentine course along the sinus wall (fig. 9). In 1 specimen the ascending (posterior) portion of the artery alone could be seen, the horizontal or final portion causing no perceptible change in the smoothness of the wall.

THE OPTIC NERVE

Projections produced by the optic nerve numbered 40. This was 13 less than those caused by the carotid artery, and it was noteworthy that

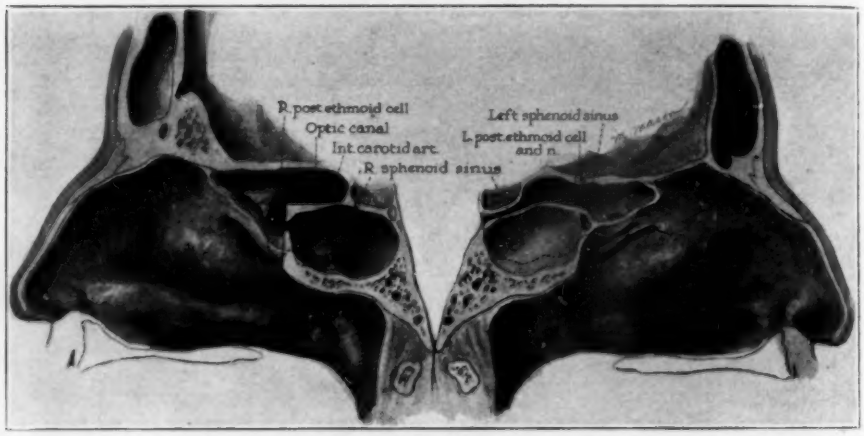

Fig. 7.—Invading posterior ethmoid cells. Large cells extend backward, crowding the sphenoid sinuses into a medial position. The left sinus in this specimen was scarcely discernible, measuring 4 by 2.5 by 5 mm. Elevations and depressions caused by nerves and blood vessels in the midcranial fossa may be observed in the lateral wall of the ethmoid cell on the right side.

all specimens containing an elevation caused by the optic nerve also contained one caused by the carotid artery (figs. 5, 7 and 9). In 5 specimens, the optic nerve canal was observed in the wall of an encroaching posterior ethmoid cell (fig. 7) rather than in the wall of the sphenoid sinus, and in 1 case, in the wall of the sphenoid sinus from the opposite side.

A consideration of the spheno-optic relations always occupies an important place in any anatomic study of the sphenoid sinus. Onodi,[9]

9. Onodi, A.: The Optic Nerve and the Accessory Sinuses of the Nose, New York, William Wood & Company, 1910.

one of the early writers on the subject, classified the relations of the optic nerve and the posterior sinuses into twelve groups, in which he found thirty-eight anatomic varieties. He called attention to the posterior ethmoid cells and the opposite sphenoid sinus as possible factors in this relationship. Mellinger [10] regarded the asymmetry of the posterior sinuses as an important factor in the vulnerability of the optic nerve. In 124 heads that he examined both optic canals were in close relation to a single sphenoid sinus in 34.1 per cent, and one optic canal was associated with both sphenoid sinuses in 23.2 per cent, while in 1 specimen the canal was related to both sphenoid cavities and a posterior ethmoid cell. Both Onodi and Mellinger and many other writers expressed the belief that the projection of the optic nerve into the sinus should be considered as a possible etiologic factor in cases of optic neuritis and other orbito-ocular diseases.

The exact cause of retinoneuritis has been the subject of many articles in ophthalmologic literature of recent years. Benedict [11] in 1933 and Lillie [12] in 1934 questioned the importance of the opticosphenoid association as an etiologic factor in retrobulbar and optic neuritis and regarded multiple sclerosis as the more likely cause. Benedict recognized the fact, however, that a rapid dissolution of symptoms takes place in many cases after the sphenoid sinus is opened, even though the cavity is apparently nonpathologic. He justified these results on the basis of the postoperative reaction, the continued congestion of the mucosa of the sinuses and adjacent tissues, the fever and the absorption of blood.

THE VIDIAN NERVE

In the present study the vidian (pterygoid) canal protruded into the sinus, causing a pronounced ridge in 36 cases and slight elevations in 12 more (figs. 5 and 6). Zuckerkandl [13] was one of the first to note that if the sinus extended into the great wing of the sphenoid, the vidian canal appeared as a ridge in the sinus floor. More recently Sluder [14] and Vail [15] made extensive clinical anatomic studies and empha-

10. Mellinger, W. J.: Optic Nerves and Their Relations in the Sphenoidal Region, Tr. Pacific Coast Oto-Ophth. Soc. **22**:85, 1937.

11. Benedict, W. L.: Retrobulbar Neuritis and Disease of the Nasal Accessory Sinuses, Arch. Ophth. **9**:893 (June) 1933.

12. Lillie, W. L.: Clinical Significance of Retrobulbar and Optic Neuritis, Am. J. Ophth. **17**:110-119 (Feb.) 1934.

13. Zuckerkandl, E.: Normale und pathologische Anatomie der Nasenhöhle und ihrer pneumatischen Anhänge, ed. 2, Vienna, W. Braumüller, 1893, vol. 1.

14. Sluder, G.: Headaches and Eye Disorders of Nasal Origin, St. Louis, C. V. Mosby Company, 1919.

15. Vail, H. H.: Vidian Neuralgia, Ann. Otol., Rhin. & Laryng. **41**:837 (Sept.) 1932.

sized the importance of the role played by the vidian nerve in clinical rhinology.

Vail stated that irritation of the vidian nerve produces the symptoms of Sluder's (sphenopalatine ganglion) syndrome. In a number of patients with this syndrome he was able to demonstrate sphenoiditis; he was also able to induce the symptoms by instillation of iodized poppyseed oil 40 per cent into the sinus. He expressed the belief that the two chief factors in the production of vidian neuralgia are (1) infection in the mucosa of a large sphenoid sinus and (2) a thin bony wall of the vidian canal, with or without dehiscence.

Sluder felt that headaches and ocular disorders with involvement of the vidian, maxillary or optic nerves are due to a hyperplastic process of the bone which narrows the foramens or canals through which the nerves pass.

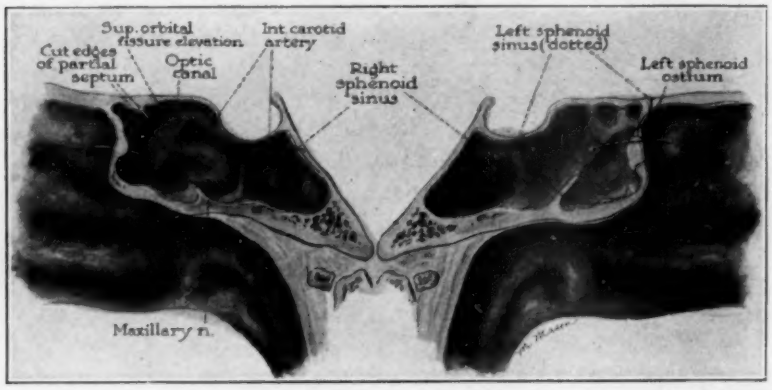

Fig. 8.—A large right sphenoid cavity which overrides its companion sinus and invades the basiocciput on both sides. The measurements of this cavity are 44 mm. in length, 23 mm. in height and 25 mm. in width.

THE MAXILLARY NERVE

The second (maxillary) division of the fifth nerve often produces an elevation in the sphenoid wall before it reaches the foramen rotundum (figs. 5, 6 and 8). This was noted in 40 of the 100 specimens, and in most of these a pterygoid recess was present which had passed below and somewhat laterally to the nerve. In most of these specimens a pterygoid (vidian) elevation was also present.

SUPERIOR ORBITAL FISSURE

The nerves which traverse the cavernous sinus and pass through the superior orbital (sphenoid) fissure often produce a bulge in the lateral sphenoid wall before reaching the orbit (figs. 5, 6 and 8). This was observed in 42 specimens; in 34 the elevation was pronounced.

Sluder [14] felt that the extent of this relationship depended to a certain extent on the size of the cavernous sinus. "If the cavernous sinus is small," he stated, "the nerve trunks may be closely associated with a sphenoid sinus even though pneumatization be limited to the body of the sphenoid bone." Sluder observed that in cases of this kind the sixth nerve, which lies lateral to the carotid artery, was somewhat removed from the wall of the sinus. When the pneumatization extends to the greater wing, the sixth nerve, along with the third, fourth and first and second divisions of the fifth nerve, is closely related to the wall of the sinus.

Houser [16] expressed a doubt whether paralysis of any nerve trunk in the cavernous sinus occurs as long as the nerve is bathed in a circulatory medium of healthy blood. He stated, however, that in a case of infection, such as exists in empyema of the sphenoid sinus, it would be quite possible for neuritis to develop with resultant paralysis in a motor nerve and sensory phenomena in a sensory trunk.

Pressure on the nerve trunks within the superior orbital (sphenoid) fissure may produce a definite syndrome. A narrowing of the margins of the fissure causes the pressure, and that may be produced by trauma, tumors, syphilis and sinal infection. Kretzschmar and Jacot [17] expressed the opinion that osteoperiostitis develops as a result of sphenoid infection and extends to involve the fissure. In a case reported by them unilateral proptosis, corneal anesthesia, paralysis of the oculomotor nerve and central scotoma developed. Complete recovery followed radical operation on the sinus.

Holt and de Rötth [18] described an "orbital apex and sphenoid fissure syndrome" which includes involvement of the optic nerve and causes a multiplicity of orbito-ocular symptoms. The condition is characterized by ptosis, diplopia and ophthalmoplegia, in which all or a part of the extrinsic muscles of the eye are involved but to a different degree. There is a wide pupil with loss of accommodation, hyperesthesia or anesthesia of the upper lid, half the forehead and the cornea and vasomotor disturbances. With these symptoms there may be pain behind the eyeball which radiates to the top of the head, forehead and temples. The narrowing of the fissure may be revealed by roentgen study. The symptoms usually clear up with removal of the cause.

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16. Houser, K. M.: Anatomic Relations of the Sphenoid Sinus to Dorello's Canal: Abducens Paralysis, Arch. Otolaryng. **16**:488 (Oct.) 1932.

17. Kretzschmar, S., and Jacot, P.: Des symptômes précoces et d'une étiologie souvent méconnue du syndrome de la fente sphenoïdale, Schweiz. med. Wchnschr. **69**:1103 (Nov. 4) 1939.

18. Holt, H., and de Rötth, A.: Orbital Apex and Sphenoid Fissure Syndrome, Arch. Ophth. **24**:731 (Oct.) 1940.

SPHENOPALATINE

The sphenopalatine elevation was marked in 30 and slight in 8 of the 100 specimens (figs. 6 and 12 *C*); in 2 more specimens it was present, not in the sphenoid, but in the floor of encroaching posterior ethmoid cells (fig. 7). This elevation serves as the roof of the pterygopalatine (sphenomaxillary) fossa (fig. 6) and, as a consequence, is in close relation to the sphenopalatine (Meckel's or nasal) ganglion which is located high up in the fossa. Further association between the fossa and the sinus is attained by the occasional pterygoid recess of the sphenoid sinus, the anterior wall of which acts as the posterior wall of the pterygopalatine fossa. The position of the ganglion in close proximity to the sphenopalatine foramen makes it subject to surface influences not only from the sinuses but from the nasal fossa as well. Sluder [14] pointed out that "characteristic disturbances follow postethmoidal and sphenoidal suppurative inflammations which cannot be explained otherwise than by assuming that the nasal ganglion has become involved by extension; some of the disturbances have been transitory, and some have persisted for many years."

The symptoms constituting the sphenopalatine ganglion syndrome, as described by Sluder, are usually preceded by coryza and start with pain at the root of the nose, in or about the eye, upper jaw and teeth. The pain extends back to the temple and the zygoma, to the ear and thence to the mastoid process and to a point 5 cm. back of the mastoid. It may also extend to the shoulder blade, the shoulder, the neck and the throat and down the arm to the fingers.

The theories of Sluder and Vail have occasioned considerable discussion in the modern literature. Many writers feel that the rationale of sphenopalatine and vidian neuralgia is founded on a faise conception, namely, that pain is a function of the autonomic nervous system, of which the vidian nerve is a member. It should be kept in mind that the great mechanism for conscious sensation in the head is the fifth nerve. It supplies the nose and sinuses and reaches the nasal cavity through fibers from its second (maxillary) division which traverse the sphenopalatine foramen with the ganglion. There is, however, no peripheral functional relation between the fifth nerve and the autonomic system. Higbee [19] maintained that cocainization and injection of the ganglion is effective because of the presence of fifth nerve fibers. Luedde [20] in a recent article cited cases of simple chronic glaucoma and spasm of accommodation in which there was recovery following anesthesia of Meckel's ganglion and other forms of intranasal therapy. The expla-

19. Higbee, D. R.: The Autonomic Nervous System: Some Fundamentals for Otolaryngologists, Ann. Otol., Rhin. & Laryng. **45**:385 (June) 1936.

20. Luedde, W. H.: The Relation of Meckel's Ganglion to Accommodation and Intraocular Tension, Illinois M. J. **79**:199 (March) 1941.

nation for these results, he said, is not found in the actual function of the sphenopalatine ganglion, but apparently depends on the accessory sensory (fifth nerve) fibers which pass through it without being a part of its true structure. Garber [21] in 1933 called attention to the fact that the maxillary nerve is in as close proximity to the sphenoid sinus as the vidian and was probably the one involved rather than the vidian in the production of the neuralgia described by Vail. One may also explain the neck and shoulder pains of Sluder's syndrome as referred impulses which pass along the descending or spinal tract of the trigeminal nerve, which extends downward to the level of the second cervical nerve. Sluder in 1912 demonstrated the permeability of the thin sphenoid wall by an intrasphenoid application of cocaine, which paralyzed the entire fifth nerve; this finding would justify Garber's contention that the maxillary nerve may be affected by irritants within the sinus. In the present anatomic study it was found that in the well pneumatized cavities both the vidian and the maxillary nerves were in close relationship with the sinus walls.

THE MANDIBULAR NERVE

The mandibular nerve (third division of the fifth nerve) was the least conspicuous of all the structures which protruded into the sphenoid walls. In only 4 cases was an elevation encountered which could be attributed to this nerve, although in 9 others the nerve and the sinus wall were in contact (fig. 6). The results in this study were similar to those of Jacob,[22] who in examining 150 heads observed that the sinus was prolonged beyond the foramen ovale in 5 for a distance of at least 5 mm. This relationship was usually found to be between the nerve and a pterygoid extension of the sinus, although in some instances no such extension was present and the relationship was established by a posterolateral prolongation of the sinus. Sluder,[14] Mosher [23] and Neivert [24] observed similar specimens, and in one of Sluder's cases "the mandibular nerve in the foramen ovale was exposed for 10 mm. to an eggshell bone separation from the sinus."

DEPRESSIONS AND RECESSES

In the well pneumatized cavities multiple elevations in the wall are to be expected. In 18 of the specimens examined all the common projec-

21. Garber, H.: Sinusitis and Neuralgia, Arch. Otolaryng. **18**:339 (Sept.) 1933.

22. Jacob, O.: Prolongement du sinus sphénoïdal creusé dans les grandes ailes du sphénoïde, Bull. et mém. Soc. anat. de Paris **75**:403-406, 1900.

23. Mosher, P., cited by Sluder.[14]

24. Neivert, H.: Morphologic Variation as a Factor in the Symptomatology of Paranasal Sinus Disease, Arch. Otolaryng. **1**:366 (April) 1925.

tions were present with depressions between them (fig. 5). The depressions themselves have certain definite characteristics, and those of one sinus closely resemble those of another. This is because the structures which cause the elevations and the depressions between them have a fairly constant location in relation to the walls of the sinus. All the depressions in the sinus walls are located adjacent to some bony structure, sphenoid or otherwise, and this permits considerable variation in the degree of extension.

Table 2 summarizes the frequency of occurrence of the various recesses.

SUPERIOR LATERAL RECESS

The recess opposite the roots of the lesser wing varies from a dimple-like depression to complete excavation of the bony process. The depression, whether extensive or not, always has a characteristic triangular

TABLE 2.—*Recesses in the Sphenoid Sinus*

Recess	Pronounced	Slight	In Lesser Wing	In Posterior Ethmoid Cell	In Opposite Sphenoid Sinus
Superior lateral (triangular)	22	14	7	4 (L. W. 2)†	1
Inferior lateral (pterygoid)	36	5	..	1	..
Anterior (palatine)	25	6	..	1	..
Superior lateral (anterior)	4	..	..	..	..
Anterior medial (septal)	5	..	..	..	..
Posterior	20	..	..	..	3

* All numbers indicate per cent.
† Lesser wing.

appearance (figs. 5, 7 and 8). The apex of the triangle points upward, reaching the roof of the sinus; its base, about 10 mm. lower in the wall, is formed by a bulge in the wall produced by the structures within the cavernous sinus. The triangle is bounded posteriorly by the groove of the internal carotid artery and anteriorly by the optic canal. This triangular depression was present in varying degrees in 41 of the 100 specimens. In 4 of these it was in an ethmoid cell, and in 1 it was in the opposite sphenoid sinus. In 9 cases of the group there was a definite projection of the sinus into the lesser sphenoid wing (fig. 5). This form of extension was noted by Coulouma and associates [25] in 2 of 16 specimens studied. They felt that in these recesses, shut off as they were from the rest of the sinus, there would be a tendency toward

25. Coulouma, P.; Bisiaux-Aufort (Mme.), and van Varseveld, F.: Le prolongement optico-alaire du sinus sphenoïdal, Echo méd. du Nord **4**:732 (Oct. 27) 1935.

retention of secretions. No clinical cases were reported to justify their contention. Retention of secretions in these recesses, though possible, is less likely to occur than in the recesses located in the sinus floor. In the present series every recess in a lesser wing communicated freely through a wide space with the main cavity of the sinus.

INFERIOR LATERAL (PTERYGOID) RECESS

Extensions into the base of the pterygoid process and the greater wing often occur between the vidian and maxillary nerves. These extensions, called inferior lateral or pterygoid recesses, vary considerably in size but resemble each other in contour (figs. 5, 6 and 8). They are usually observed as oval hollowed-out recesses in the floor and lateral wall of the sinus. They are the largest of the sphenoid extensions and as a rule average approximately 15 mm. in the transverse dimension and 20 mm. anteroposteriorly. The width is regulated largely by the relative position of the vidian and maxillary nerves. In small heads the width is less than in the larger ones because the two nerves lie nearer to each other. In length, however, no such structures control the expansion, and the excavation may be so pronounced as to bring the sinus into contact with both the mandibular nerve posteriorly and the maxillary sinus anteriorly (fig. 6). The pterygoid depression was pronounced in 36 per cent of the present series, and this frequency varies but slightly from the findings of Eadie,[26] who observed similar recesses in 30 per cent of 137 postmortem specimens, and those of Cope,[4] who found the condition present in 40 per cent of his 289 specimens. Neivert [24] found only 11 (5 per cent) pterygoid recesses in 220 sets of sinuses. This is an unusually low percentage but probably included only the pronounced type and those with extensive pneumatization of the greater sphenoid wings.

These hollowed-out depressions in the lateral portion of the floor of the sinus offer perfect reservoirs for the collection of secretions and as such add considerably to the importance of the sphenoid cavity in clinical rhinology and ophthalmology. A case illustrating this importance was reported by Elles: [27] A man, 20 years old, with bilateral accommodation paralysis and unilateral scotoma obtained complete recovery within an hour after operation on his sphenoid sinuses. Roentgen studies had previously revealed a pterygoid extension, which at operation was found to be filled with a yellowish fluid. A horizontal partition (partial septum) blocked the drainage from the infected area.

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26. Eadie, C. M.: Posterior Nasal Sinusitis, M. J. Australia **1**:487 (March 12) 1938.

27. Elles, N. B.: Bilateral Accommodation, Paralysis and Unilateral Scotoma in Sphenoidal Sinus Disease, Am. J. Ophth. **21**:1365 (Dec.) 1938.

The following case is one of chronic sphenoiditis in which a pterygoid recess was also revealed by roentgen ray examination.

REPORT OF A CASE

V. O., a white doorman, was seen, Jan. 26, 1933, complaining of headaches of several years' duration. The pain would start in the frontal area and then extend to the top of the head, back to the occiput and down the arms. When he had these attacks, he was weak and could work only with difficulty. He also complained of postnasal discharge with a morning accumulation of crusts in his nasopharynx.

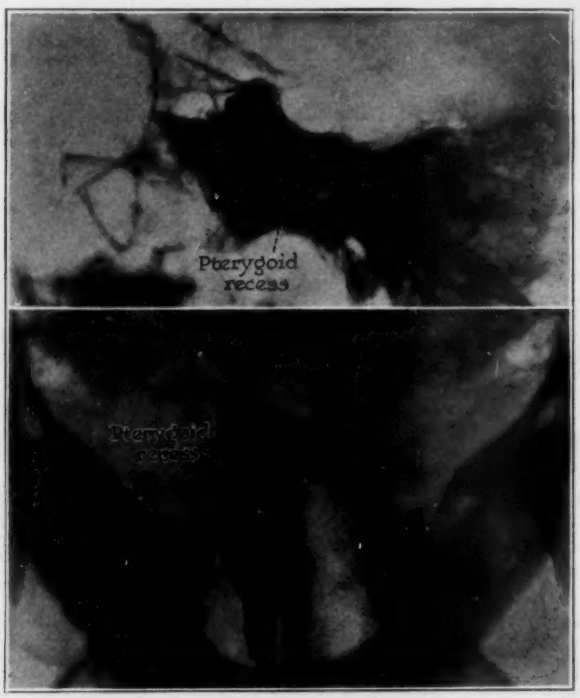

Fig. 9.—Roentgenograms of sinuses in case 1, showing extensive pneumatization of the right sphenoid sinus with pterygoid recess.

Four months previously the patient had received irrigations of both antrums, which helped but little. Another rhinologist had told him a radical operation on his sinuses would be necessary. Examination revealed a nasal septum highly deviated to the left with bilateral atrophy of the ethmoid turbinates. The right sphenoid ostium was visible on anterior rhinoscopy, and crusts were present in that area.

Roentgen study showed most of the sinuses to be involved in an infective process, and films made with iodized poppyseed oil 40 per cent showed a large right sphenoid sinus with a pterygoid recess (fig. 9).

The maxillary sinuses were normal on irrigation. Purulent material was removed from the right sphenoid sinus by lavage through the ostium. The patient showed little improvement until the opening was enlarged and several thick crusts

were washed out of the sinuses. Since then he has had only an occasional return of the headache, and this disappears with irrigation of the sinuses. The crusts have disappeared from the nose and nasopharynx, and he is rarely uncomfortable.

The collection of thick crusts in the sphenoid cavity is a common characteristic of sphenoiditis, according to Ridpath.[28] Undoubtedly, deep recesses in the sinus floor predispose toward such accumulations.

ANTERIOR EXTENSION

The intimate relationship often present between the sphenoid and maxillary sinuses was noted by Mosher [29] in 1903, and at that time he described his transantral approach to the sphenoid. Such an operation naturally has a limited scope and is applicable only in those few cases in which the walls of the two cavities come together. In the others the intervening pterygopalatine fossa with the contained nerves and blood vessels renders the technic impracticable. Contact between the two cavities was observed in 18 specimens; in 2 of these a sphenoid bulge was present in the antral wall (fig. 6). In 3 of the specimens the sphenomaxillary contact was made between a lateral pterygoid recess and the posterior antral wall, and in 14 it was brought about by a laterally extending palatine recess which reaches the medial wall of the antrum. In the remaining specimen, recesses in both the pterygoid and palatine areas made contact with the posterior antral wall, the sinuses coming together for a distance of 23 mm.

Extensions from the sphenoid into the palatine and ethmoid bones (anterior recesses) pass over the sphenopalatine elevation to invade the ethmoid field and occasionally reach the orbital wall (figs. 6 and 12 *C*). When the anterior extension of the sinus is high and lateral, a recess may be formed anterior to the optic nerve (fig. 8), and this combined with the triangular depression posterior to the nerve produces an almost complete encirclement of the optic canal.

POSTERIOR EXTENSIONS

Posteriorly, the sinus may invade the basilar part of the occipital bone, passing around the ascending portion of the internal carotid artery and bringing this vessel into marked relief in the sinus wall (fig. 8). Recesses dorsal to the carotid artery were observed in 23 specimens, in 3 of which the sphenoid sinus from the opposite side occupied the area. In these, both the internal carotid arteries, although occupying their normal positions in the cranium, were outlined in the walls of a single wide sphenoid sinus (fig. 8). Occasionally the sinus extends upward to

28. Ridpath, R. F.: The Sphenoid Sinus, Laryngoscope **44**:657 (Aug.) 1934.

29. Mosher, H. P.: The Anatomy of the Sphenoid Sinus and the Method of Approaching It from the Antrum, Laryngoscope **13**:177-215 (March) 1903.

invade the posterior clinoid process, and in some instances excavations in both the anterior and the posterior clinoid processes cause a pronounced semilunar projection of the hypophysial fossa into the lumen of the sinus (fig. 10).

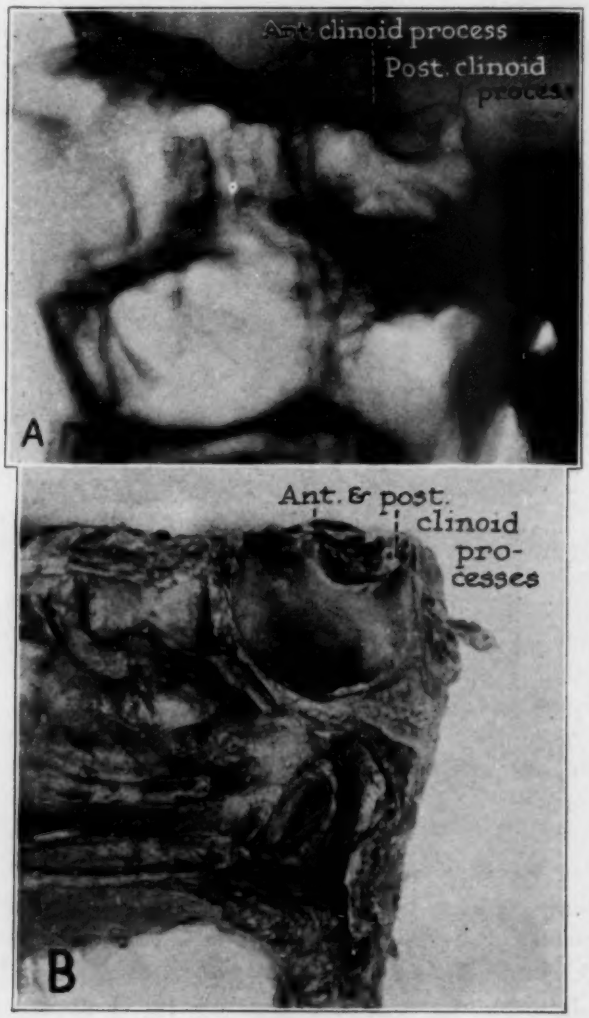

Fig. 10.—Excavation of the clinoid processes. *A*, roentgenogram of a lateral view, showing excessive pneumatization in both the anterior and the posterior clinoid process. *B*, photograph of a specimen showing a similar expansion. In cases of this type there is a pronounced semilunar projection of the hypophysial fossa into the lumen of the sinus.

The sella turcica (hypophysial fossa) is commonly in close relation to one or both sphenoid sinuses. The exceptions to this are the poorly

pneumatized cavities of the conchal and presphenoid type and those in which the sinuses have been displaced by invading ethmoid cells. Dixon [30] reported similar findings in his series of 1,600 skulls and found several specimens with the sella separated from the sinus by a bony wall 10 mm. thick. In the majority, however, the pituitary gland is separated from the sphenoid, at least in its anterior portion, by only a very thin wall and the sinus mucosa. Teed [31] collected from the literature many reports of pituitary gland involvement in suppurations of this region. In 10 cases with meningitis and 6 without, the gland was infected or completely destroyed. In 1 of his own cases the gland was reduced to a suppurative mass. Disturbances of pituitary function from disease of the sphenoid sinus have been noted, including a few cases of pituitary syndrome and some of glycosuria, which developed subsequent to the onset of the sinusitis.

OSSEOUS GROWTHS IN THE SINUS WALLS

Spurs projecting from the walls of the sinus were comparatively rare in the series of specimens under investigation. In 2 specimens a spur was present in the posterior wall of each sphenoid sinus. One of these protruded into the lumen of the sinus for a distance of 7 mm. In 1 specimen an exostosis 5 mm. long jutted from the floor of a pterygoid recess, and in another a small bony growth was present on the lateral wall opposite the maxillary nerve.

Most of the partial septums were found in a certain area and were similar in size and shape. Such a septum gave the impression of being a shelf on the posterior wall, with a concave anterior edge which slanted downward to the floor medially (figs. 5 and 13). Laterally, it reached a point below the posterior root of the lesser wing, slightly anterior to the internal carotid artery. It is near this point that in the development of the bone the two wings join the body of the sphenoid with a confluence of three planes of fusion. The partial septum is thought by most observers to begin to form as the growing sinus contacts the compact bone of this area. It is usually first seen about the tenth year [2] as a ridge on the posterior sinus wall (fig. 3 *B* and *C*). As the wall retreats, the bony plate elongates backward accordingly.

A partial septum was present in 41 of the 100 specimens. In 21 of these, it was attached to the sinus floor and the posterior and lateral walls. In 13 of the 21 specimens it extended the entire width of the sinus from the floor at its junction with the medial wall to a point high

30. Dixon, F. W.: A Comparative Study of the Sphenoid Sinus, Ann. Otol., Rhin. & Laryng. **46**:687 (Sept.) 1937.

31. Teed, R. W.: Meningitis from the Sphenoid Sinus, Arch. Otolaryng. **28**: 589 (Oct.) 1938.

on the lateral wall. In 6 others a vertically placed septum extended transversely across the sinus, causing a posterior pocket in the floor of the sinus. In a few specimens the septum was attached to the medial wall and roof of the cavity, and in 2 an anteriorly placed septum formed a shelf a few millimeters above the sphenoid ostium.

The clinical significance of the partial septum in the sphenoid sinus is readily understood, especially in those cases in which the bony plate is so placed that it acts as a barrier to sinus drainage.[27]

THICKENED MUCOSA

In the 13 specimens lined with a thickened mucous membrane, 7 contained a partial septum; 6, however, were without a septum, and 4 of

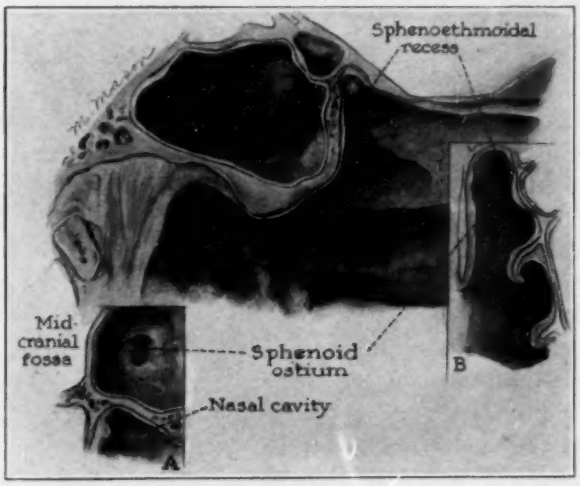

Fig. 11.—Thickened mucosa in a sphenoid sinus with involvement of the ostium and the sphenoethmoid recess. The sinus, though large, contained no irregularities in its bony walls.

these contained no irregularities whatever in their walls. In 1 specimen with no septum, elevation or depression to disturb the even contour of its walls, a thickened mucosa was present not only throughout the sinus but in the sphenoethmoid recess as well, and the sphenoid ostium was almost completely closed by the process (fig. 11).

DEHISCENCES

Dehiscences in the walls were not observed in the gross examination of the 100 specimens, although such defects have been noted by numerous investigators both in the anatomic and in the clinical field. The importance of perforations in the sphenoid walls as a cause of intracranial complica-

tions is questionable. According to Teed,[31] most writers refer to dehiscences solely as anatomic anomalies and think that infection is more likely to reach the cranium by way of the perforating vessels which pass directly from the mucosa of the sinus through the bone to the dura.

ENCROACHMENT

A thorough understanding of the relationship of the sphenoid sinuses to each other is possible only when both halves of a skull are examined. In the present investigation 11 skulls were so studied, and in these a pronounced asymmetry of the two cavities was noted. In only 3 of the 11 could the sphenoid sinuses be remotely considered as symmetric. In these, a comparatively thick septum in the medial plane separated the two cavities. In the other 8, a thin septum, although in the midline anteriorly, deviated to one side or the other in its dorsal portion. Among the 100 specimens many were found wherein one sphenoid cavity had almost completely taken over the area of the other (figs. 8 and 10). In many cases of this type the crowded sinus occupied but little space in the body of the sphenoid bone, its main cavity being located in the palatine or pterygoid area.

One marked case of counterencroachment was observed. The anterosuperior part of the left sphenoid sinus extended to the right beyond the midline, while posteriorly the right sphenoid sinus occupied the entire postsphenoid area.

Encroachment by posterior ethmoid cells was noted in 12 cases, and encroachment by ethmoid cells from the opposite side, in 5. In 3 of the 5 there was double encroachment, the invading cell being accompanied by the opposite sphenoid sinus as well. In 2 cases a small sphenoid sinus was overridden by a posterior cell from the same side and by the sphenoid sinus from the opposite side, and in 1 skull both sphenoid cavities were overridden by a single posterior ethmoid cell which drained into the left supreme meatus (Van Alyea,[32] fig. 10).

AGENESIS OF THE SPHENOID SINUS

No cases of complete absence of the sinus were observed. In 1 skull (fig. 7) a large posterior ethmoid cell had invaded the sphenoid bone on each side, causing lateral recesses and forcing the sphenoid sinuses into a medial position. The left sphenoid sinus in this case was scarcely discernible; it was a diminutive cavity measuring 4 by 2.5 by 5 mm. and was almost entirely filled in with mucous membrane. Schaeffer [7] also encountered similar rudimentary cavities and felt that agenesis is an uncommon finding. Hajek [33] stated that the sinus may be entirely absent

32. Van Alyea, O.: Ethmoid Labyrinth, Arch. Otolaryng. **29**:881 (June) 1939.

33. Hajek, M.: Pathology and Treatment of the Inflammatory Disease of the Nasal Accessory Sinus, St. Louis, C. V. Mosby Company, 1926, vol. 1.

in cases of deficient resorption of the body of the sphenoid bone. In such cases a small depression in the anterior wall of the body of the sphenoid bone lined by an extension of the nasal mucous membrane is present instead of the ostium sphenoidale. Davis [2] found no agenesis in his series of 114 heads in various stages of development.

THE SPHENOID OSTIUM

The sphenoid ostium is usually oval, with the long axis in a near vertical plane. The average diameters in the 100 specimens with the mucosa intact were 3.3 and 2.25 mm. The largest ostium was 8 mm. high and 5 mm. wide but had the appearance of having been enlarged by operation. Another large opening measured 5.5 by 5 mm.; 1, 6 by 4 mm.; 4, 6 by 3 mm., and 4 more 5 by 4 mm. The most common size was 3 by 2 mm.; this occurred in 20 per cent of the specimens. Four were extremely small, being 1.5 mm. in height by 1 mm. in width. Dixon [34] in an examination of 1,600 skulls noted that the average diameter of ostiums of the round type was 3 mm., while the average dimensions of the oval ostiums were 4 by 2.4 mm.

The sphenoid ostium is located in the anterior wall of the sinus and opens into the sphenoethmoid recess. Measurements were taken to determine its relation to the sinus roof and floor, to the cribriform plate and the nasal septum and to the floor of the nose and the inferior nasal spine.

In 34 specimens (approximately one third) the opening was equidistant from the roof and floor of the sinus. In approximately one half (52 per cent) it was nearer the roof than the floor, while in the remaining 14 specimens it was nearer the floor. In 1 extremely unusual case the opening was in the roof facing downward (fig. 12 *D*), and in 3 others the openings were just below the roof. All 3 were cases in which upward growth of the sinuses had been blocked by the presence of other cavities. In the entire group studied the average distance from the roof was 7.7 mm. and from the floor 10.7 mm.

THE SPHENOETHMOID RECESS

The cribriform plate, which covers the olfactory fissure and sphenoethmoid recess, lies in a lower horizontal plane than the superior wall of the sphenoid sinus. The sphenoid ostium, as a consequence, was nearer by approximately 3 mm. to the cribriform plate than to the sinus roof. The average distance from the nearest edge of the opening to the roof of the sphenoethmoid recess in the present series was 4.1 mm. and to the nasal septum 2.2 mm. In many specimens the opening of the sphenoid sinus extended from the septum to the lateral wall of the

34. Dixon, F. W.: Catheterizing the Sphenoid Sinus, Arch. Otolaryng. **30**:994 (Dec.) 1939.

sphenoethmoid recess, thereby occupying the entire width of the nasal portion of the anterior wall of the sphenoid sinus (fig. 12 *C*). In other instances the orifice was small and located in the depths of the recess, 5 to 7 mm. from the septum (fig. 12 *B*). The average width of the recess itself was 4.8 mm., the measurement being taken from the septum

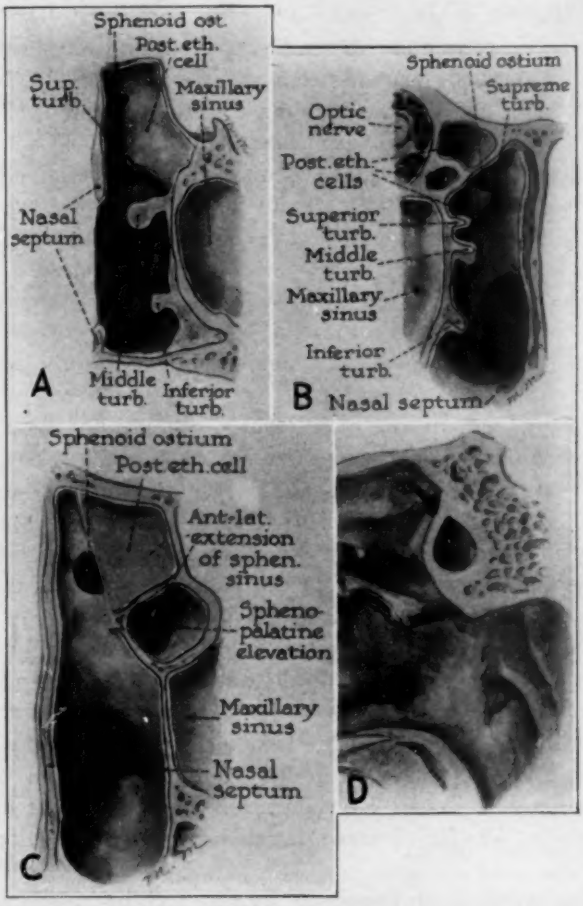

Fig. 12.—Types of sphenoid ostium: *A*, average type. The orifice shown measures 3.5 by 2.5 mm. It is located in a shallow sphenoethmoid recess, is less than 2 mm. from the nasal septum and is so placed as to lend itself easily to probing. *B*, small ostium, deep in the recess, difficult to locate. *C*, large opening, typical location, easily probed. *D*, ostium in roof of sinus. This type of opening would be difficult to engage with the cannula tip.

to the lateral nasal wall above the attachment of the most superior turbinate.

The distance from the center of the ostium to the floor of the nasal cavity was the most constant of all the measurements taken, averaging

37.5 mm. There were few extremes; 1 measured 50 mm., and 1, 30 mm., but most frequently (72 per cent) the range was between 35 and 40 mm.

The distance from the ostium to the inferior nasal spine averaged 58 mm., ranging in the majority of cases between 55 and 66 mm., with extremes measuring 47 and 70 mm.

CLINICAL ACCESSIBILITY OF THE SPHENOID OSTIUM

The sphenoid ostium may be probed without difficulty in the majority of cases. Anatomic characteristics which occasionally hamper the approach to the ostium are: (1) a deviated nasal septum, (2) a narrow olfactory groove, (3) a narrow sphenoethmoid recess, (4) a small ostium, (5) a deeply placed ostium and (6) an overhanging ethmoid cell. Any one of these structural characteristics adds to the probing difficulties, while a combination of two or more may render the orifice entirely inaccessible.

In 50 of the specimens the ostium almost completely replaced the posterior wall of the sphenoethmoid recess. In these cases the medial edge of the opening came within a millimeter or two of the nasal septum, and its lateral edge reached the lateral wall of the recess (fig. 12 *A* and *C*). In this type of case a cannula tip approaching the anterior wall of the sphenoid sinus could scarcely miss the ostium.

In the remaining 50 specimens the aperture was more laterally placed, its nearest margin being 3 mm. or more from the septum. In these cases a search might be required before the ostium would be encountered. In 16 of these specimens, aside from the lateral position of the aperture, anatomic barriers were present which hampered the approach to the sinus. In this group of 16 specimens the ostium was entirely inaccessible or to be reached only after prolonged search. In 11 of the group a small ostium laterally placed was the cause of the probing difficulty. In 3 others the recess was too narrow to permit the passage of any but a small cannula, and in 1 of these an additional barrier was an overhanging posterior ethmoid cell. In 2 specimens the recess was not only narrow but the opening was far removed, occupying a lateral position in a deep recess.

The configuration of the nasal septum and its relationship to the ethmoid turbinates should be considered in an analysis of the accessibility of the sphenoid ostium. In the present investigation, however, this was not possible, for the septum had been removed from most of the specimens prior to examination. Dixon,[34] on the other hand, examined skulls with the septum intact. He was able to enter only 70 per cent of 600 sinuses studied. Other investigators found the ostium more accessible. Sewall [35]

35. Sewall, E. C.: Cytologic Study of Diseases of the Sphenoid Sinus, Ann. Otol., Rhin. & Laryng. **42**:391 (June) 1933.

in a series of 110 specimens was able to enter the sphenoid ostium in all but 12. He maintains that a cannula may be placed in the ostium with great regularity on exercise of a little care, patience and practice.

SPHENOID CANNULA

The preferred type of cannula is thin and slightly flexible with a blunt end (fig. 13). Its over-all length is 12.5 cm., which is several centimeters shorter than most instruments used in the approach to the sphenoid sinus. The shaft of the cannula is 9 cm. long, with a marker 7 cm. from the tip. The length of 9 cm. is deemed adequate for all cases; for no specimen was observed in which the sphenoid ostium was a greater distance than 8 cm. from the anterior part of the nares. The distal portion of the cannula is bent in a smooth curve; this places the tip 4 mm. lateral to the shaft. At the shoulder of the cannula a wire marker protrudes at an

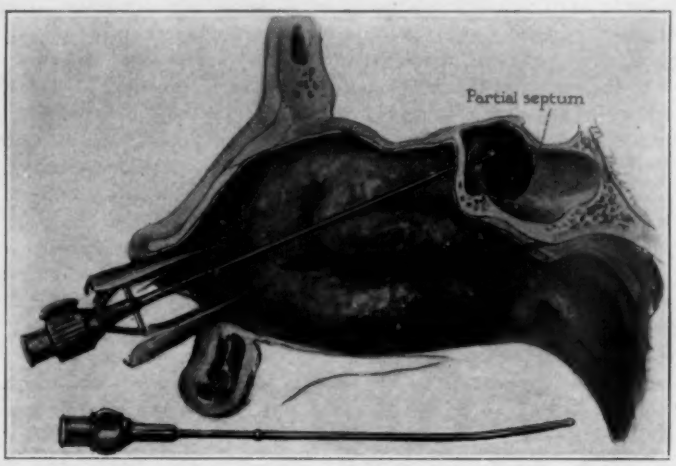

Fig. 13.—Sphenoid cannula in place. At the shoulder of the cannula, wire markers protrude from the shaft at an angle of 23.5 degrees. The cannula should be passed with the lower marker parallel with the floor of the nose. This brings the upper marker parallel with the dorsum of the nose. The distal portion is bent into a smooth curve which places the tip 4 mm. laterally to the shaft.

exact angle of 23.5 degrees. This attachment serves as a guide in determining the proper angle of approach to the ostium. The measurements used in computing this angle were 37.5 mm., the average distance from the ostium to the nasal floor and 68 mm., the average distance to a point 1 cm. above the floor in the anterior part of the nares. In skulls which are larger the distance is greater, but as a rule proportionately so, and the angle changes but little. The same rule applies to the smaller skulls, and rarely does the angle vary more than 3 or 4 degrees. If the cannula is passed with the marker parallel to the nasal floor and held 1 cm. above the floor, it will point directly at the ostium in the vast majority of cases.

TECHNIC OF CANNULIZATION

With the region under local anesthesia, the cannula should be passed into the olfactory fissure with its concavity facing the convex nasal surface of the middle

and superior turbinates. As the cannula tip passes into the sinus the edge of the ostium may be palpated. The experienced rhinologist, as a rule, senses the contact thus made and knows that the sinus has been reached. In his paragraph on sounding the sphenoid sinus, Hajek [33] said, "The sensation of having slipped through a relatively narrow opening into a cavity is a very definite and distinct one." If the operator is not conscious of this sensation with the tip at a depth of 80 mm., he must assume that the tip is well within the lumen of the sinus. If he is uncertain of this, he may withdraw the cannula and attempt to ascertain the location of the sinus by probing at some other point.

Roentgenograms are of considerable aid in determining the position of the anterior wall. These are of particular value when lateral views are taken with probes or cannulas placed at various points in the olfactory fissure. The same view also determines the location of the posterior wall, which is important when a puncture is to be made through the anterior wall. Should the natural opening prove inaccessible, puncture of the anterior wall is the recognized procedure. This may be performed near the midline with a reasonable degree of safety. The dangers associated with this operation are puncture of the lateral wall of the sinus with injury to the internal carotid artery, the cavernous sinus or other structure adjacent to the wall and puncture through the roof of the sinus or the cribriform plate anterior to the sinus. It should also be kept in mind that because of a tendency toward asymmetry the sinus from the opposite side may be the one entered. Attention was directed to this possibility by Watson-Williams,[36] who reported several cases of sphenoiditis with ocular involvement in which a large overriding cavity hampered the search for the infected sinus. Pressman [37] reported a similar case, one of left iritis in which at first the large right sphenoid sinus was unwittingly opened through the left nasal cavity. The position of the cannula tip was determined by roentgen study, which also revealed an infection in a small laterally placed left sinus. The iritis cleared up after irrigation of the left sphenoid sinus. For puncture of the anterior wall a cannula such as that designed by Tremble [38] carries certain safety elements which may well be incorporated in all instruments to be used for this purpose. The tip of the cannula is sharp and points downward. At a point 11 mm. back of the tip a vertical tongue acts as a guard to prevent the instrument from penetrating too deeply into the sinus. Previous roentgen study and exercise of a reasonable amount of care should eliminate the possibility of mishap during irrigation, whether the procedure is carried out through an artificial or through the natural opening.

SUMMARY

The sphenoid sinus originates during the third fetal month as an invagination of the nasal mucosa in the posterior extremity of the cartilaginous nasal capsule. The developmental stages through which the sinus passes are regulated somewhat by changes which take place earlier during the development of the sphenoid bone itself.

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36. Watson-Williams, P.: Chronic Nasal Sinusitis and Its Relation to General Medicine, Baltimore, William Wood & Company, 1930.

37. Pressman, J. T.: Iritis Caused by Asymptomatic Sphenoiditis with Anomaly of the Sphenoid Sinuses, Arch. Otolaryng. **26**:83 (July) 1937.

38. Tremble, G. E.: Irrigation of the Sphenoid Sinus, Arch. Otolaryng. **32**: 952 (Nov.) 1940.

Accumulation of dense bone at certain of the planes of fusion accounts for arrested development of the sinuses and for any encroachment of one sinus on the territory of the other. The growth of the sinuses in some cases is not halted at these planes, and they may become large thin-walled cavities intimately related to adjacent structures. Expansion may continue in these cases to the formation of recesses in adjacent bony processes, sphenoid or otherwise.

The frequency of the occurrence of recesses and other anatomic characteristics was determined by a study of 100 specimens from cadavers. The clinical significance of the anatomic relations thus established has been considered.

The average dimensions of the sinus were obtained, as well as those of the sphenoid ostium.

The ostium was also studied as to its location in the sphenoethmoid recess and as to its clinical accessibility. The opening was deemed accessible in a vast majority of cases. In 16 per cent, largely because of its size and location, the ostium was inaccessible or could be reached only with difficulty.

A cannula has been designed with a curved tip and with markers protruding from the shaft at an angle of 23.5 degrees, which facilitates the search for the sphenoid opening.

Thickened mucous membrane indicative of sinal infection was noted in the gross examination of 13 of the 100 specimens. In 10 of these a similar thickening was present in the mucosal lining of the sphenoethmoid recess, and in all of the 10 cases the opening was completely or almost completely blocked by adhesions or thickened mucosa.

PRIMARY CARCINOMA OF THE EXTERNAL AUDITORY CANAL AND MIDDLE EAR

REVIEW OF THE LITERATURE; REPORT OF A CASE OF CYSTIC ADENOID EPITHELIOMA (BROOKE'S TUMOR) OF THE EXTERNAL AUDITORY CANAL

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The problem of cancer is one of vital interest to clinician and pathologist alike. Close cooperation between the two has added a wealth of knowledge to the subject within relatively recent years. Mutual interest is aroused by any unusual clinical manifestation or variation in microscopic structure. It is largely as a result of such mutual interest and close cooperation that much progress has been made in a combined attack on this strange malady.

Malignant growth of the middle ear and external auditory canal is one of the rarest lesions that the otologist is called on to treat. The paucity of authentic case reports in the literature speaks for the relative uncommonness of this type of otitic disease. Many modern textbooks give no more than passing mention to the condition. The statistical reports of large aural clinics bear witness to its rarity. The average otologist spends a lifetime in practice without seeing more than 1 or 2 growths of this nature. In order to obtain a comprehensive picture of this subject, it is essential to study the literature with some care. In the past an early diagnosis has been the exception rather than the rule, and the results of therapeutic procedures have left much to be desired. It is encouraging to note, however, that today the diagnosis is being made at an earlier stage than formerly and that recent case reports show a longer average survival after treatment than was common a few years past.

Wilde and Roudot, Schwartze, Lucae, Kidd and others in the period around 1775 were among the first to discuss carcinoma of the middle ear. Politzer,[1] in his textbook on diseases of the ear, written in 1883,

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From the Department of Otolaryngology of the Eye, Ear, Nose and Throat Hospital.

1. Politzer, A.: Text-Book of Diseases of the Ear, translated and edited by J. P. Cassells, Philadelphia, Henry C. Lea's Son & Company, 1883.

devoted several pages to a surprisingly good and accurate clinical description of malignant disease in this region. Zeroni,[2] in an exhaustive review of the literature, found 121 otitic tumors recorded between 1804 and 1899. His personal experience was limited to 2 cases of primary malignant disease of the tympanum. In the interval between 1899 and 1917 Newhart [3] was able to find reports of 34 cases of carcinoma of the middle ear. This number includes several cases mentioned in discussions but not reported in extenso. Only 8 cases were reported in the American literature. In an endeavor to learn something of the incidence of the disease in this country and to gain, if possible, some information as to the ratio of carcinomas diagnosed to those reported, he sent letters to leading otologists throughout the country. At the time his article was printed, he had received replies from 247 of these. Of this number 192 had never encountered a case of malignant disease of the middle ear. Forty-five otologists reported a total of 51 cases. Two had 5 cases each. Only 2 of these 51 cases had been recorded in the literature. In a thorough search of the literature Yates [4] found 14 cases listed between 1917 and 1924 and 24 more between 1924 and 1936. He reported an extremely interesting instance of alveolar carcinoma complicated by aneurysm of the carotid artery and by subperiosteal abscess.

Among 212,000 cases of aural disease encountered in the hospital and dispensary of the Manhattan Eye, Ear and Throat Hospital from 1905 to 1924, Robinson [5] could find only 48 diagnosed as tumor, giving a ratio of 1 to 4,000. Story [6] reported that a case of malignant disease of the ear had not been seen in the largest aural hospital in Ireland in seventeen years. Bezold [7] found only 4 malignant growths of the ear in 20,000 otologic cases; 1 growth affected the middle ear and 3 the external ear. From 1906 to 1929, of 646 patients seen in private practice in whom the external acoustic meatus was involved, Fraser [8] found malignant disease of the external meatus in only 3. Of 6,605 cases of

2. Zeroni, cited by Furstenberg, A. C.: Primary Adenocarcinoma of the Middle Ear and Mastoid, Ann. Otol., Rhin. & Laryng. **33**:677, 1924.

3. Newhart, H.: Primary Carcinoma of the Middle Ear: Report of a Case, Laryngoscope **27**:543, 1917.

4. Yates, E. C.: Primary Carcinoma of the Middle Ear, Kentucky M. J. **34**:501, 1936.

5. Robinson, G. A.: Malignant Tumors of the Ear, Laryngoscope **41**:467, 1931.

6. Story, cited by Burton, F. A.: Epithelioma of the Middle Ear and Mastoid: Report of a Case, Laryngoscope **27**:755, 1917.

7. Bezold, cited by Risch, O., and Lisa, J. R.: Primary Carcinoma of the External Auditory Canal and Meatus: Review of the Literature and Report of a Case, Laryngoscope **48**:668, 1938.

8. Fraser, J. S.: Malignant Disease of the External Acoustic Meatus and Middle Ear, Proc. Roy. Soc. Med. (Sect. Otol.) **23**:1235, 1930.

disease of the external ear and meatus encountered in the department of otology of the Royal Infirmary, Edinburgh, Scotland, 13 were found to be cases of malignant disease.[9] Connal[10] mentioned 4 carcinomas and 2 sarcomas among 15,000 patients seen in the General Ear Hospital in Glasgow, Scotland. McBride[11] encountered only 3 cases of malignant disease of the ear in twenty years at the Edinburgh Infirmary, Edinburgh, Scotland, and in private practice. Junod[12] said that only 6 primary cancers of the middle ear had been seen in over twenty-five years at the Basel, Switzerland, ear clinic, among 45,000 patients with aural disease. Furstenberg[2] found only 2 primary malignant growths of the middle ear among 40,000 patients admitted to the department of otolaryngology at the University of Michigan Medical School. One of these was a hemangiosarcoma and the other an adenocarcinoma. In reviewing the records of the twelve year period previous to 1935 Schall[13] found that only 15 patients out of 90,040 with pathologic conditions of the ear seen at the Massachusetts Eye and Ear Infirmary (a ratio of 1 to 6,000) had neoplasms of the middle ear. In discussing Whitehead's paper Tod[14] quoted the ten year records of the London Hospital, where 200,000 cases are seen annually. During this time only 1 cancer of the middle ear was seen. West and Bonner have the unique distinction of having seen several such lesions within a relatively short time. West[11] saw 7 in two years at St. Bartholomew's Hospital, London, England, 6 of which gave evidence of having arisen in the middle ear. Bonner[15] saw 5 or 6 within a few years.

As to the causation of primary cancer of the middle ear and external auditory canal, otologists still are without definitely established scientific facts. Among leading otologists throughout the country there is a wide diversity of opinion as to the relative significance of the various etiologic factors. Most are agreed, however, that chronic irritation is of major

9. Maxwell, D.: Analysis of Statistical Tables, J. Laryng. & Otol. **44** (pt. 1): 105, 1929.

10. Connal, cited by Risch, O., and Lisa, J.: Primary Carcinoma of the External Auditory Canal and Meatus: Review of the Literature and Report of a Case, Laryngoscope **48**:668, 1938.

11. McBride, P., in discussion on West, C. E.: Four Cases of Carcinoma of the External Auditory Meatus, Proc. Roy. Soc. Med. (Sect. Otol.) **2**:34, 1908-1909.

12. Junod: Primary Cancer of the Middle Ear, Schweiz. med. Wchnschr. **52**: 510, 1922; abstracted, J. A. M. A. **79**:413 (July 29) 1922.

13. Schall, L. A.: Neoplasms Involving the Middle Ear, Arch. Otolaryng. **22**:548 (Nov.) 1935.

14. Tod, in discussion on Whitehead, A. L.: A Case of Primary Epithelioma (?) of the Tympanum Following Chronic Suppurative Otitis Media, Proc. Roy. Soc. Med. (Sect. Otol.) **1**:34, 1907-1908.

15. Bonner, A.: Epithelioma of Cutaneous External Meatus, Tr. Otol. Soc. U. Kingdom **2**:39, 1900-1901.

importance as a contributory factor. In spite of the frequently observed clinical relationship and in spite of many years of experimental laboratory research, this postulation still remains largely on a theoretic basis. One cannot help noting, however, the frequent association of cancer in this region with chronic purulent otorrhea, which is present in fully 75 per cent of the cases reported. Newhart [8] expressed the belief that epithelium invades the middle ear from the external auditory canal and that as a result of the trauma incident to the chemical irritation of a chronic purulent discharge a carcinoma develops.

The age incidence is similar to that of carcinoma involving other parts of the body. The disease is more common in persons in the fourth decade, but instances have been recorded among much younger persons. Alexander saw a carcinoma in a boy of 17; Leidler reported one in a patient 17 years of age; Nager one at 19, and Knapp one at 20.[3]

The sexes seem to be equally affected.

Heredity plays the same part in cancer in this region as in cancer of other body structures.

Trauma, excluding that resulting from surgical procedures, cannot reasonably be considered a serious factor, as the anatomic situation of the external auditory canal and of the middle ear makes them relatively impervious to the influence of gross traumatic injury. There are a few cases on record in which the author has attempted to show a relationship between trauma and the origin of a new growth. From our study of case histories we were not impressed with the importance of this relationship.

The following may rightfully be considered as predisposing factors: (1) chronic ulcers of a specific or a nonspecific nature, (2) intractable dermatitis, (3) sebaceous cysts, (4) papilloma and (5) chronic suppurative mastoiditis with granulations and polyps in the external canal.

The origin of carcinoma of the middle ear has been a matter of debate, and the issue still remains somewhat confused. One of the difficulties hitherto encountered has been that patients have usually been observed when the disease was in a relatively late phase and the ear canal, middle ear and mastoid filled with cancerous tissue, with extension to the surrounding structures. Under such circumstances the origin could not be definitely determined clinically, and consequently the matter resolved itself into a series of postulations. Fortunately, today patients are seen when the disease is at an earlier stage, so that more opportunity is provided for close clinical observation and study. It has been proposed by some that the cancer originates in the external auditory meatus, with extension inward to involve the structures of the middle ear. Other observers believe that the process originates deep in the external auditory canal or in some cases from the tympanic mem-

brane, with secondary involvement of the middle ear. Barnes [16] said that it is probable that a large number of cancers originate in the lining of the mastoid cavities, which has become altered during a prolonged suppuration. In these cases there are no symptoms which can properly be called "early," since at this time the mastoid is already full of malignant tissue. Under such circumstances the symptoms which first attract the attention of the patient are pain and facial paralysis, which at the onset is partial. According to Panse, a malignant neoplasm that originates in a closed tympanic cavity probably results from a metaplasia of the cylindric cells of the lining membrane to the squamous cell type or has its origin from the glandular elements within the cavity.

The disease runs a rapid course. In the earlier cases reported a life expectancy of one to two years after the onset of symptoms was usual. More recent reports on therapy sound an encouraging note, with mention of patients living as long as six or seven years after early institution of appropriate therapy. A typical history embraces chronic otorrhea of years' duration, associated with granulations that are friable, bleed easily on manipulation and tend to recur on removal, followed by the development of pain, facial paralysis and, in the late stages, involvement of the neighboring structures, with death from such conditions as toxemia, exhaustion and cachexia.

As to symptoms, several facts stand out clearly when one examines the case histories recorded in the literature. There is usually a discharge, which may be foul and purulent or bloody or both. Some foreign observers separate malignant growths in the region of the middle ear into two groups, those associated with chronic otorrhea and those in which it is absent.

Bleeding may be in the form of a blood-stained discharge or in the form of a free hemorrhage following manipulation or surgical intervention. The latter is difficult to arrest.

Pain is an early symptom. It is unusually severe and at a relatively early stage requires morphine sulfate for relief. Dundas-Grant said that the characteristic feature of the early stage, when the disease in the meatus is comparatively inconspicuous, is the intense pain, which radiates over wide areas and which is out of proportion to the amount of pathologic change evident in the ear. On the other hand, Bowman [17] took exception to the opinion that pain is an early manifestation of cancer of the external auditory canal. He pointed out that the size of the growth indicates the stage of advancement when the patient first

16. Barnes, E. B.: Carcinoma of the Ear, Proc. Roy. Soc. Med. (Sect. Otol.) **23**:1231, 1930.

17. Bowman, R. J.: Carcinoma of the External Auditory Canal, Middle Ear and Mastoid, Ann. Otol., Rhin. & Laryng. **49**:225, 1940.

applies to the physician, who is often misled by the brief duration of symptoms, which usually begin with the onset of pain. There are different theories as to the cause of pain. Barnes [16] stated the belief that pain is due to an infection of the tumor mass and of the skin lining the external acoustic canal. The simultaneous occurrence of purulent discharge and pain in a number of cases reported seems to lend some support to this contention. Other authors have expressed the belief that pain is due to an early invasion of the perichondrium and cartilage or to those as yet unknown qualities which are responsible for pain associated with cancer in other parts of the body. Whatever the cause may be, it is a definitely established clinical fact that the pain is severe and a source of distraction to the patient.

Granulations are usually present and show a tendency to friability and free bleeding on manipulation.

Facial paralysis usually appears at some time during the course of the disease, and in some cases it may appear early. In 2 cases reported by Barnes [16] the onset of facial paralysis was simultaneous with the appearance of pain, and in 3 cases in which it was an early sign it was only partial for a long time. In cases reviewed by Risch and Lisa [7] facial paralysis followed within a few weeks to a year after the onset of otorrhea.

Tumor as a presenting symptom is found occasionally.

Cachexia is common and is due to the disturbance created by pain and to the toxins produced by the neoplasm itself.

Deafness is proportionate to the degree of involvement of the hearing mechanism from obstruction, inflammation or actual invasion by tumor tissue. Symptoms referable to disturbance of the static labyrinth may appear at any time during the course of the disease. In some cases a relatively high degree of hearing may be maintained even in the late stages.

The late symptoms are those accruing from an invasion of the surrounding structures. The parotid gland and the temporomandibular joint are involved at a relatively early stage. Bone is destroyed, with the formation of sequestrums as a result of pressure and the obliteration of blood supply. There is invasion of the postauricular soft tissues. The lateral sinus may be surrounded and obliterated, and in the final stages the meninges, the brain and the cranial nerves may be involved. Erosion of the internal carotid artery with fatal hemorrhage has been observed. The dura and the labyrinthine capsule are resistant to invasion. The regional lymph nodes are frequently enlarged, but the presence of cancer cells is rare. On microscopic section chronic inflammatory changes may be seen. The preauricular and deep anterior

cervical nodes are the first to become involved. Guttman [18] emphasizes the early involvement of the regional lymphatic system. Metastasis to distal body organs has been seen but is unique.

The problems encountered in the diagnosis of cancer in this region are virtually the same as those found in investigating cancer in other organs of the body. Bowman [17] has rightfully stressed the importance of a careful history, stating that frequently symptoms of minor character have been found to be present fully a year before the development of the major symptoms, which caused the patient to seek medical attention. In a series of 70 cases analyzed by Scott [19] only 19 patients presented themselves with symptoms which immediately aroused suspicion of malignant disease. A careful inspection of the ear, nose and throat is essential, and a complete physical examination and laboratory investigation are always in order. In the final analysis, biopsy is the only recourse for an accurate scientific diagnosis. We cannot emphasize too much the necessity of this procedure in any case in which the history and the physical examination are in any wise suggestive of malignant disease. Scott and Mollison [20] have recently pointed out that ulcerative lesions of the external auditory canal which grossly simulated malignant growths have been found on pathologic section to be of a benign and nonspecific character. These have responded completely to local treatment. Roentgenograms are of little value in the early phases but may be of inestimable value in determining extension to surrounding parts. It is only by a thorough attack on cancer in this region at a relatively early stage that otologists can hope to bring the treatment of otitic cancer up to the levels of treatment found in the management of cancer of other organs. The characteristic marked obscurity of symptoms will defeat that purpose unless otologists become cancer conscious.

The prognosis is poor. According to reports in the more recent literature, cures of six to seven years are being obtained when adequate therapy has been instituted early. This is in contrast to the earlier reports, in which the life expectancy after the onset of symptoms was given as about eighteen months.

As to treatment, one can say immediately without fear of contradiction that there is no compromise with cancer in the region of the

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18. Guttman, M. R.: Primary Carcinoma of the Tympano-Mastoid with Report of a Case, Illinois M. J. **56**:150, 1929.

19. Scott, P., in discussion on Malignant Disease of the Ear (Excluding the Pinna), Proc. Roy. Soc. Med. (Sect. Otol.) **32**:1087, 1938.

20. Scott, S., and Mollison, W. M.: Some Cases of Non-Malignant Ulceration in the External Auditory Meatus Resembling the Appearance of Early Malignant Disease, J. Laryng. & Otol. **54**:350, 1939.

external auditory canal and the middle ear. The surgical procedures must be most radical. A thorough radical mastoidectomy, with removal of as much of the diseased parts as is consistent with a safe operative procedure, must be done. This must be followed by adequate postoperative irradiation directed by a skilled roentgenologist who has had experience in treating malignant disease in this region. Ullman [21] called attention to the fact that in his experience the cartilage of the ear recovers well from large doses of radiation. Pfahler and Vastine [22] reported favorable results in the treatment of cancer of the external auditory canal, middle ear and mastoid region. For lesions in the external auditory canal they recommended the taking of a specimen for biopsy, with destruction of the remaining tissue by electrodesiccation or curettage followed by the application of a capsule of radium of sufficient strength to destroy the surface disease. For cancer of the middle ear and mastoid they advised high voltage roentgen therapy. The conscientious surgeon will not deny his patient the advantage of thorough operative procedure followed by proper irradiation. Either alone is not sufficient. Above all, it must be remembered that each patient must be given individual treatment.

A relatively wide variety of histologic types of malignant growth have been recorded as involving the external acoustic canal and middle ear. This list includes squamous cell, basal cell and basal-squamous cell types, epithelioma adenoides cysticum (Brooke's tumor), endothelioma, hemangioendothelioma and lymphoendothelioma, sarcoma, fibrosarcoma and myxosarcoma, neurofibroma, adenocarcinoma and carcinoma of the ceruminous glands. In this connection should be mentioned dermoids, because of their frequent association with malignant disease.[23] Tyler [24] described malignant degeneration of a parotid cell nest in the external auditory canal. Williams and Pastore [25] reported the first case of neurofibroma of the facial nerve with destruction of the mastoid and bony labyrinth. Lukens [26] reviewed the literature and

21. Ullman, H. J.: Treatment of Ear Malignancies, California & West. Med. **37**:369, 1932.

22. Pfahler, G. E., and Vastine, J. H.: The Treatment of Cancer in the Region of the Ear, Am. J. Roentgenol. **37**:350, 1937.

23. Marshall, G. G.: Dermoid Teeth in the External Auditory Canal, with Comment on Teratomas and Dermoids in General, New England J. Med. **214**: 202, 1936.

24. Tyler, A. F.: Malignant Degeneration of a Parotid Cell Nest in the External Auditory Canal, Nebraska M. J. **10**:431, 1925.

25. Williams, H. L., and Pastore, P. N.: Neurofibroma of the Facial Nerve in the Facial Canal: Destruction of the Labyrinth and Mastoid Process, Arch. Otolaryng. **29**:977 (June) 1939.

26. Lukens, R. M.: Adenocarcinoma of the External Auditory Canal, Ann. Otol., Rhin. & Laryng. **45**:567, 1936.

found only 4 cases of adenocarcinoma of the external auditory canal reported prior to 1936. He reported the fifth case. The large preponderance of histologic types falls into the squamous and basal cell groups, with a decided majority of the squamous cell type. Broders [27] analyzed a series of 63 cases of epithelioma of the ear and found the squamous cell type present in 62 per cent, the basal cell type in 33 per cent and the melanotic variety in 2 per cent of cases. In his series the auricle was involved in 85 per cent, the external auditory canal in 14 per cent and the middle ear in 2 per cent of cases. Risch and Lisa [7] collected from the literature 35 cases published prior to 1938, in which primary cancer of the external auditory canal and meatus was not associated with chronic otorrhea, and added a case of their own. In 1933 Nichinsson [28] reported a case of epithelioma adenoides cysticum of the external auditory canal and stated that no report of a similar case could be found in the literature available up to that date.

Our interest in malignant disease in this region was quickened by the recent observation of a cystic adenoid epithelioma (Brooke's tumor) of the external auditory canal. To our knowledge the case of Nichinsson [28] (1933) and the case recorded in this paper are the only ones in which Brooke's tumor involving the external auditory canal, middle ear or mastoid region has ever been reported. In Scott's case 2, a pathologic diagnosis was returned of a "tumor having characteristics intermediate between those of rodent ulcer with cystic change and a sebaceous adenoma."

Since the almost simultaneous description of the neoplasm by Brooke [29] and by Fordyce,[30] in 1892, there has been much discussion in the literature concerning the classification of Brooke's tumor and its relation to other epithelial tumor growths. Summerill and Hutton,[31] in an excellent review of the literature on this subject, pointed out the comparative rarity of the tumor and emphasized the fact that it can be distinguished histologically from syringocystadenoma, adenoma sebaceum, cylindroma, and adenoma of the sweat glands, with which it is frequently confused both clinically and pathologically. Molesworth,[32] in an excel-

27. Broders, A. C.: Epithelioma of the Ear: A Study of Sixty-Three Cases, S. Clin. North America **1**:1401, 1921.

28. Nichinsson, A. G.: A Case of Epithelial Tumor of the External Auditory Canal, Monatschr. f. Ohrenh. **67**:210, 1933.

29. Brooke, H. G.: Epithelioma Adenoides Cysticum, Brit. J. Dermat. **4**:269, 1892.

30. Fordyce, J. A.: Multiple Benign Cystic Epithelioma, J. Cutan. Dis. **10**: 459, 1892.

31. Summerill, F., and Hutton, J. G.: Multiple Benign Cystic Epithelioma (Epithelioma Adenoides Cysticum), with Summary of Literature, Arch. Dermat. & Syph. **26**:854 (Nov.) 1932.

32. Molesworth, E. H.: Rodent Ulcer, M. J. Australia **1**:878, 1927.

lent article on rodent ulcer, emphasized the extreme rareness of Brooke's tumor as contrasted with the everyday frequency of rodent ulcer in Australia.

According to Ewing,[33]

> the structure varies but is dominated by the presence of embryonal epithelial cells arranged in cords, masses, and alveoli, and of groups of concentric hornifying epithelium inclosing homogeneous material. The adenoid structure appears as fingerlike projections from the basal epithelium or ducts of glands, and they commonly contain minute or large cystic areas of hyaline, colloid, or mucoid material. The cells enclosing these foci are often cylindrical. The squamous cells in concentric form undergo extensive hornification or calcification, and these structures may compose the bulk of the tumor (Schopper). Or the structure of reticulated epithelioma, resembling rodent ulcer with many thin cell strands, may be present (Csillag, Wolters).
>
> The origin of the tumor is referred by Brooke to the normal cells, or to embryonal cell groups derived from the hair follicles (lanugo), and from the basal cells of the epidermis. While this view is supported by most recent observers, Torok and Darier derived their tumors from the sweat glands and W. Pick from the sebaceous glands. The identity of these structures with Brooke's tumor is doubtful but it seems probable that in different cases the origin may involve different elements with corresponding variations in structure.

Clinically, these tumors occur as small pea-sized to bean-sized round firm nodular painless translucent lesions, most frequently about the pinna, nose, cheek, forehead, scalp, chest and back and relatively uncommonly below the pelvis. They frequently appear about the age of puberty and have a predilection for females. There seems to be some hereditary tendency. The growth is slow over a period of years, and ulceration is rare. These tumors are potentially malignant and may metastasize, although this is unusual. They differ from rodent ulcer in their occurrence in younger patients, failure to ulcerate and predilection for females.

REPORT OF A CASE

L. H., a Negress aged 32, came to the clinic of the Eye, Ear, Nose and Throat Hospital on July 1, 1940, complaining of pain in the right ear.

About three months before the patient's admission to the clinic a sharp excruciating pain that radiated deep into the ear developed in the right auditory canal. It was intermittent and became worse during the period before admission. There were no associated symptoms.

Past Medical History.—The patient stated that pain in the right ear first developed in the latter part of 1937 and that she had been treated in the clinic previously for a "growth in the ear." Our clinic record is incomplete with respect to treatment during that period. A notation shows that a "papillary growth" in the external canal was touched with 50 per cent silver nitrate in April and in May 1938 and that the growth was painful when touched. The patient was not heard of again until September, when she returned to the

33. Ewing, J.: Neoplastic Diseases, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

clinic and, with the region under local anesthesia, had a "small tumor" of the right external canal removed. Unfortunately, this specimen was not sent to the laboratory for pathologic study. A note made on the clinic record in May 1939 is to the effect that there was a recurrence of the growth and that removal was advised, but this was not done, as the patient disappeared from the clinic and was not heard of again until she came under our observation on July 1, 1940.

The patient had mumps in childhood but prior to the illness concerned here had never had any serious illnesses or operations. Her average weight was 136 pounds (61.7 Kg.); on admission her weight was 142 pounds (64.5 Kg.). She had six children, all of whom were in good health. The menses were regular and she was without disability during the menstrual period. She had never had any miscarriages. She had been doing daily housework for years. During the past two or three years she had been subject to attacks of occipital and bitemporal headache associated with pain in the back of the neck. She stated that she had a constant postnasal discharge of mucoid character, occasional dry cough and frequent head colds and attacks of sore throat.

Family History.—The mother was living and well. There was no family history of cancer, tuberculosis, diabetes or other significant disease on the maternal side. The paternal history was not known.

Social History.—The patient used coffee, tobacco and alcohol in moderation.

Physical Examination.—The nasal mucosa was pale and thickened, especially over the posterior tips of the middle and inferior turbinates bilaterally, as seen on nasopharyngoscopic examination. The septum was deflected. The pharynx was granular. The tonsils were small and embedded and the anterior pillars injected. The laryngeal mucosa was pale and slightly thickened. Both tympanic membranes showed some thickening and retraction. The audiogram showed a 20 decibel loss in the left ear and a 30 decibel loss in the right. The Rinne test was positive. Weber's test showed lateralization to the right ear, and the Gellé test was positive. The eustachian tubes were patent. The left external auditory canal was normal in appearance. On the right side a small firm round sessile tumor was seen occupying the medial half of the cartilaginous portion of the canal and situated chiefly on the posterior and inferior walls. The tumor was covered with skin and measured roughly 5 by 5 mm. There was no ulceration or evidence of secondary infection in the tumor mass proper or in the skin lining the external auditory canal. The mass was exquisitely tender to palpation with a probe. No granulations were present. The tympanic membrane was intact. The caloric tests showed normal function of the labyrinths. The frontal sinuses and the antrums were clear on transillumination. There was extensive pyorrhea, and a small firm protuberance sprang bilaterally from the medial surface of the mandible below the second premolars. The pupils, extraocular movements and fundi were normal. The heart, chest and abdomen were normal. The reflexes were active. The blood pressure was 125 systolic and 75 diastolic. Roentgenograms of the sinuses and mastoids failed to reveal any pathologic condition.

Laboratory Findings.—The total red cell count was 4,800,000, the value for hemoglobin 80 per cent and the color index 0.8+. The total white cell count was 5,500, with 63 per cent neutrophils and 37 per cent small lymphocytes. There were 4 per cent each of myelocytes and juvenile forms and 2 per cent stab forms. The urine showed a specific gravity of 1.010 and an acid reaction. The urine was normal chemically. Microscopic examination of urinary sediment showed pus cells and epithelia. The Wassermann and the Kahn test of the blood were negative.

Operation and Course.—On July 27, 1940, with the patient under anesthesia induced by 1 per cent procaine hydrochloride and epinephrine, one of us (J. C. P.) extirpated from the external auditory canal on the right side a tumor mass approximately 5 mm. in diameter. A "sleeve" of tissue that included the membranous and cartilaginous canal was removed. There was no gross evidence of infiltration of the underlying tissues, and the bony external canal was grossly healthy. Moderately free bleeding was encountered, which was easily controlled by packing with iodoform gauze. The wound was allowed to granulate from the bottom. Recovery was uneventful. The patient was seen last on Nov. 6, 1940. There was no evidence of recurrence or of stenosis of the canal. At the time of writing she is being referred for postoperative irradiation.

Pathologist's Report (G. H. H.).—Gross Examination: "The tumor measures 8 mm. in width and 5 mm. in depth, and the surface is covered by skin. It is raised above the surrounding edge of healthy tissue, is smooth in outline and shows no ulceration."

Microscopic Examination: "The tumor consists of epithelial masses, separated by connective tissue and showing glandular arrangement and cyst formation. It extends downward from just beneath the epidermis, and no direct connection with the basal layer can be determined definitely. The tumor structure consists of masses of small hyperchromatic basal cells, some of which are in sheets and others in glandular arrangement, with numerous small and some larger cysts, containing mucoid material. Many of the cells lining the cysts are flattened out. Only a few mitotic figures are present."

SUMMARY

We have endeavored to review the literature on primary carcinoma of the middle ear and external auditory canal and have included a report of a case of cystic adenoid epithelioma (Brooke's tumor) of the external auditory canal. In making this study we have been impressed with several outstanding facts. Carcinoma in this region is relatively rare. We are of the opinion that it is somewhat more common than our examination of the literature indicates. Apathy on the part of otologists in reporting known cases of malignant disease, together with failure to make a diagnosis in a certain number of cases, has undoubtedly denied the literature an appreciable number of case reports. This perhaps is far more true of the past era than of the present. We cannot emphasize too much the necessity for a careful taking of the history, physical examination, laboratory study and, above all, biopsy. In the final analysis a histologic study establishes the diagnosis of a benign or a malignant lesion. The causation still remains largely a question of theoretic considerations. This disease runs a rapid course, with characteristic invasion of the neighboring structures in the terminal stages. The most frequent histologic types are of the squamous or basal cell variety. The characteristic features of the clinical course are chronic otorrhea, pain that is out of all proportion to pathologic involvement and notably severe, and granulations that are friable, bleed easily and tend to recur, associated with facial paralysis, which usually occurs at some time during the course of the disease and which may frequently be an

early manifestation. The cause of the pain associated with cancer in this area is not known. In the absence of any gross or microscopic infection, ulceration or discharge, we are led to conclude that the severe pain in the case reported by us was due either to an involvement of the perichondrium and cartilage or to those unknown qualities responsible for the pain of cancer in other organs. The treatment of cancer in this area has not been on the same high plane as treatment of cancer in other superficial parts of the body, but, fortunately, present day trends in treatment are destined to bring therapeutic results up to a more satisfactory level. So far as we have been able to determine from the literature available to us, only 1 case of Brooke's tumor of the external auditory canal has been recorded previously in the literature. We are adding a second report. Owing to the confusion which has existed in the pathologic classification of this histologic type, it seems probable that other cases may have been recorded under a different diagnosis. In many respects the disease in our case manifested the general characteristics of Brooke's tumor as it occurs generally over the body and at the same time showed the usual characteristics of malignant growths situated in the external auditory canal, being unique chiefly in its relative rareness and site of occurrence. It was present in a female at the fourth decade of life. The course was slow, and the growth recurred after removal. Pain was marked and out of proportion to the pathologic condition, and there was no ulceration or involvement of the regional lymph nodes. We hope that our comments will serve to direct attention to the possibility of cancer in this region and will also stimulate the otologist to report such cases as come under observation.

Dr. Spencer Wood and Dr. R. M. Street gave us permission to report the case of cystic adenoid epithelioma of the external auditory canal, from their services in the Department of Otolaryngology of the Eye, Ear, Nose and Throat Hospital, New Orleans. Drs. F. E. LeJeune and Philip Bayon made helpful comments.

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PARALYSIS AND PARESIS OF THE VOCAL CORDS

A STATISTICAL REVIEW

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Dysfunction of the vocal cords is ascribed to several recognized causes, such as lesions of either the central or the peripheral nerves or local changes, such as neoplasm or ankylosis of the various joints, particularly of the cricoarytenoid joint. The dysfunction may be partial (paresis) or complete (paralysis) and, whether present in one or both cords, may or may not be manifested clinically. It is well known that, particularly if a nonfunctioning vocal cord lies in the cadaveric position, the opposite cord will often cross the midline, so that no symptoms either in phonation or in respiration result. If one vocal cord lies in the midline, the compensatory function of the other cord is assured. Both cords lying in the cadaveric position permit the patient to carry on respiration with little embarrassment, but phonation is poor; however, the reverse is true if both cords lie paralyzed in the midline.

Since Semon [1] first formulated his theory of proclivity of the abductor fibers to the adductor fibers, in 1881, much has been published regarding the anatomy and physiology (both normal and abnormal) of the larynx and the causes of diseases which affect this structure. The complexity of the problem speaks for itself. The present study was undertaken to reemphasize a few points in regard to paralysis and paresis of the vocal cord and to present some cases of unusual etiologic interest.

PREVIOUS STATISTICS

In 1891 Avellis, as pointed out by Permewan,[2] published a report of a series of cases of unilateral paralysis of the recurrent laryngeal nerve. In approximately 50 per cent of the cases of this series, the cause of the paralysis was unknown.

1. Semon, F.: On the Proclivity of the Abductor Fibres of the Recurrent Laryngeal Nerve to Become Affected Sooner Than the Adductor Fibres, or Even Exclusively, in Cases of Undoubted Central or Peripheral Injury or Disease of the Roots or Trunks of the Pneumogastric Spinal Accessory, or Recurrent Nerves, Arch. Laryng. **2**:197-222 (July) 1881.

2. Permewan, W.: Discussion on the Aetiology of Unilateral Paralysis of the Recurrent Laryngeal Nerve, Proc. Roy. Soc. Med. (Sect. Laryng.) **6**:147-154 (May) 1913.

Hall [3] reported 116 cases of laryngeal nerve paralysis, including a series of his own and cases collected from the Proceedings of the Section of Laryngology of the Royal Society of Medicine, 1893 to 1907. In 82 cases the left cord, and in 33 the right cord, was involved.

Sendziak, quoted by Delavan,[4] reported 1,017 cases of paralysis of the vocal cords. He divided these into cases of paralysis of the abductor fibers, of which there were 526 and cases of paralysis of the recurrent nerve, of which there were 491.

In 1910 Guder and Dufour, quoted by Permewan,[2] reported 63 cases of unilateral paralysis of the laryngeal nerve. In 36 cases the left side was affected and in 27 the right side.

New and Childrey [5] in 1932 reported 217 cases of paralysis of the vocal cord in which the causation was known. In an additional 105 cases, the cause was unknown.

In 1933 Smith, Lambert and Wallace [6] reported 235 cases of paralysis of the vocal cord.

According to the many studies recorded in the literature up to the present, the most common factors causing paralysis of the vocal cord are aortic aneurysm, tumor of the mediastinum, tumor of the esophagus and hypopharynx, goiter and lesions originating in the central nervous system.

BASIS OF PRESENT STATISTICAL REVIEW

The 257 cases of paralysis and paresis of the vocal cord encountered at the University Hospital, Ann Arbor, Mich., from 1925 through the first half of 1938 have been reviewed. Of this group there was complete paralysis of the vocal cord in 212, while in the remaining 45 there was paresis of the cord.

SEX AND AGE INCIDENCE

Approximately 69 per cent of the males with paralysis of the vocal cord were found in the age group of 40 to 70 years. Sixty-eight per cent of the females were between 30 and 60 years old. This is readily explained by the higher incidence of goiter at a younger age in females. With paresis the same parallel was shown in the females but not in the males.

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3. Hall, F. DeH.: Discussion on the Aetiology of Unilateral Paralysis of the Recurrent Laryngeal Nerve, Proc. Roy. Soc. Med. (Sect. Laryng.) **6**:139-143 (May) 1913.

4. Delavan, D. B.: The Etiology of Paralysis of the Recurrent Laryngeal Nerves of Peripheral Origin, Ann. Otol., Rhin. & Laryng. **17**:631-644 (Sept.) 1908.

5. New, G. B., and Childrey, J. H.: Paralysis of the Vocal Cords: A Study of Two Hundred and Seventeen Medical Cases, Arch. Otolaryng. **16**:143-159 (Aug.) 1932.

6. Smith, A. B.; Lambert, V. F., and Wallace, H. L.: Paralysis of the Recurrent Laryngeal Nerve: A Survey of Two Hundred and Thirty-Five Cases, Edinburgh M. J. **40**:344-354 (July) 1933.

SYMPTOMS

Except in textbooks, symptoms resulting from dysfunction of the vocal cord have been little stressed. Every laryngologist has encountered a patient with a negative subjective history only to be surprised on objective examination of the larynx to find functional impairment of the vocal cord.

The symptoms, in their order of frequency, are as follows:

	Paralysis (Percentage)	Paresis (Percentage)
No symptoms	30	50
Hoarseness	43	29
Weakness and tiredness of the voice	10	4
Dyspnea	8	12
Strangling on foods	6	4
Laryngeal stridor	1	0

It is significant that one-third to one-half the patients in each of the two groups have no symptoms.

CAUSATION

Even with the methods of diagnosis in use at present, it is not always possible to find a cause for functional impairment of the vocal cord. As stated previously, in approximately 50 per cent of Avellis'[2] series of cases the cause was unknown. New and Childrey[5] encountered, in addition to their 217 cases in which the etiologic factors were known, 105 in which the cause was undetermined.

In the series reported here the impairment in approximately 22 per cent of the cases of paralysis (table 1) and in 22 per cent of the cases of paresis (table 2) was of unknown causation. It will be seen that cases of impairment following thyroidectomy were included. If these are excluded, the percentage of instances in which the cause of impairment is unknown rises to 37 in the paralytic group and 32 in the paretic group—figures that compare with those of New and Childrey.[5] As seen in tables 1 and 2, in most of the cases there was an obvious cause. However, certain of these cases are of particular interest, and I should like to point them out.

A CASE OF PARALYSIS OF CONGENITAL ORIGIN

An infant boy 6 weeks old had had periods of stridor and periods of cyanosis since birth. Laryngeal examination showed the cords fixed in the midline. Respirations became so embarrassed while the patient was under observation that tracheotomy was resorted to. Three days later he died of severe tracheobronchitis. He had associated spina bifida occulta and meningocele of the sacral region.

TABLE 1.—*Paralysis of the Vocal Cords*

Cause	Male	Female	Right Cord				Left Cord				Bilateral				Grand Total
			Median	Cadaveric	Unspecified	Total	Median	Cadaveric	Unspecified	Total	Median	Cadaveric	Unspecified	Total	
Unknown	32		1	3	7	11	4	2	11	17	..	..	4	4	
		16	2	1	1	4	2	1	7	10	1	..	1	2	48
Congenital lesion	1		..	..	..	..	..	..	..	..	..	..	1	1	1
Bulbar paralysis		1	1	..	..	1	..	..	..	..	..	..	..	..	1
Amyotrophic lateral sclerosis	1		..	..	1	1	..	..	..	..	..	..	..	..	1
Progressive spinal muscular atrophy		1	..	..	..	..	1	..	..	1	..	..	..	..	1
Syringomyelia and syringobulbia	4		1	..	..	1	1	..	1	2	1	..	..	1	
		1	..	..	..	..	..	1	..	1	..	..	..	..	5
Thrombosis of the posterior inferior cerebellar artery	4		1	..	..	1	2	..	1	3	..	..	..	..	
		1	..	..	..	..	..	..	1	1	..	..	..	..	5
Central nervous system syphilis	2		..	..	..	..	..	..	..	..	..	..	2	2	
		1	..	..	..	..	..	..	..	..	..	..	1	1	3
Miscellaneous group of central nervous sytem origin	3		..	..	..	..	2	1	..	3	..	..	..	..	
		2	..	..	1	1	..	1	..	1	..	..	..	..	5
Polioencephalitis		1	..	..	..	..	..	..	1	1	..	..	..	..	1
Tumors of the hypopharynx and the esophagus	7		..	..	2	2	2	..	..	2	..	1	2	3	
		1	..	..	..	..	1	..	..	1	..	..	..	..	8
Nongoitrous tumors of the neck	2		..	..	1	1	..	..	..	..	1	..	..	1	
		2	..	..	1	1	..	..	1	1	..	..	..	..	4
Benign goiter	3		2	..	1	3	..	..	..	..	..	..	..	..	
		6	2	1	2	5	..	..	1	1	..	..	..	..	9
Malignant goiter	3		1	1	..	2	..	..	1	1	..	..	..	..	
		2	..	..	..	..	2	..	..	2	..	..	..	..	5
Exophthalmic goiter		1	1	..	..	1	..	..	..	..	..	..	..	..	1
Postoperative thyroidectomy	10		..	..	2	2	..	2	3	5	2	1	..	3	
		74	4	11	14	29	11	11	12	34	7	1	3	11	84
Postoperative pneumonectomy	1	..	..	..	..	..	..	..	1	1	..	..	..	..	1
Aortic aneurysm	4		..	..	..	..	..	1	3	4	..	..	..	..	
		1	..	..	..	..	1	..	..	1	..	..	..	..	5
Apical fibrotic tuberculosis	3		..	..	..	..	1	..	2	3	..	..	..	..	
		3	..	..	3	3	..	..	..	..	..	..	..	..	6
Mitral stenosis		2	..	..	..	..	..	..	2	2	..	..	..	..	2
Malignant condition of the mediastinal gland	8		1	..	..	1	2	..	5	7	..	..	..	..	
		4	1	..	2	3	..	..	1	1	..	..	..	..	12
Inflammation of the mediastinal glands		1	..	..	..	..	..	1	..	1	..	..	..	..	1
Undetermined type of tumor of the mediastinal glands	1		..	..	..	..	..	..	1	1	..	..	..	..	1
Congenital heart disease	1		..	..	..	..	1	..	..	1	..	..	..	..	1
Enlarged thymus		1	..	1	..	1	..	..	..	..	..	..	..	..	1
Total	90	122	18	18	38	74	33	21	55	109	12	3	14	29	212

CASES OF PARALYSIS OR PARESIS DUE TO LESIONS IN THE CENTRAL NERVOUS SYSTEM

As pointed out by Furstenberg,[7] cortical lesions are not capable of producing complete laryngeal paralysis unless they are large enough to affect the cortical bulbar fibers on both sides, in which event the paralysis is bilateral and is of the spastic type. Nuclear lesions (nucleus ambiguus) or infranuclear lesions produce a flaccid type of paralysis, unilateral if one nucleus is involved and bilateral if both nuclei or infranuclear pathways are involved.

Table 1 presents 22 cases, in which the lesions were considered to be of nuclear type or immediate infranuclear type, and table 2, 11 cases in which the lesions were found to be of the supranuclear type producing

TABLE 2.—*Paresis of the Vocal Cords*

Cause	Sex		Right Cord	Left Cord	Bilateral	Total (Cause) ♂ and ♀
	Male	Female				
Unknown	7		3	4	..	
		3	1	2	..	10
Bulbar paralysis	1		..	..	1	1
Amyotrophic lateral sclerosis	2		..	..	2	2
Pseudobulbar palsy	1		..	1	..	1
Miscellaneous group of central nervous system origin	6		3	1	2	
		1	..	1	..	7
Nongoitrous tumors of the neck	2		2	..	..	2
Benign goiter	2		2	1	1	
		3	..	..	1	5
Malignant goiter		1	..	..	1	1
Postoperative thyroidectomy	2		1	1	..	
		12	1	9	2	14
Aortic aneurysm	1		..	1	..	1
Mitral stenosis	1		..	1	..	1
Total	25	20	13	22	10	45

paresis. In the 22 cases of table 1, the paralysis may be considered to be of the flaccid type, while in the 11 cases of table 2 it may be considered to be of the spastic type. In discussing cortical lesions, New and Childrey [5] twice noted "an unusual type of incomplete paralysis"; in the first instance this was probably caused by arteriosclerosis of the central nervous system, and in the second it was probably due to polioencephalitis. The 11 cases of paresis in this series are no doubt of the same order.

A number of points in the cases of dysfunction of central origin analyzed in tables 1 and 2 should be emphasized:

1. Of the 2 patients with bulbar paralysis, 1 had complete paralysis of the right vocal cord and the other had bilateral paresis of the cords.

7. Furstenberg, A. C.: An Anatomical and Clinical Study of Central Lesions Producing Paralysis of the Larynx, Tr. Am. Laryng., Rhin. & Otol. Soc. **41**: 141-146, 1935.

In other words, at the time the latter patient was examined, the lesion was in an early stage and the progression in both medullary nuclei was incomplete.

2. Two patients with amyotrophic lateral sclerosis had bilateral paresis of the vocal cords and another had paralysis of the right vocal cord.

3. The progressive spinal muscular atrophy noted in 1 patient was of the Dejerine-Sottas type. The left vocal cord lay paralyzed in the midline.

4. Syringomyelia and syringobulbia accounted for 5 cases of paralysis of the vocal cords. One of the patients, a man, had bilateral abductor paralysis of the vocal cords, necessitating tracheotomy and submucous resection of the cords. Another, a girl aged 7, presented scoliosis and malformations of the spine originating from the nervous system, myelodysplasia of the lumbosacral cord associated with syringobulbar paralysis of the twelfth and ninth cranial nerves on the left and paralysis of the left vocal cord.

5. Thrombosis of the posterior-inferior cerebellar artery also accounted for 5 cases of paralysis of the vocal cord. In 4 of these the left side was affected and in the fifth the right. There were 4 male patients and 1 female. The portions of the medulla affected by the thrombosis were the dorsolateral retro-olivary area, the lateral spinothalamic tract, the reticulospinal tract and the area of the nucleus ambiguus. In all these cases sympathetic changes were noted by hypohydrosis of the face, neck and arm and sometimes of the leg on the same side as the lesion.

6. In cases in which syphilis of the central nervous system was the cause only nuclear types of lesions were noted, and all of these were bilateral. In 1 case there was bilateral involvement of the superior laryngeal nerve as well as of the recurrent laryngeal nerve. There was loss of sensation about the hypopharynx and larynx as well as complete loss of motion of the vocal cords. These were lying in a flaccid bowed position. The patient was seen on two different occasions at a two year interval. There was no change in the laryngeal picture or in his symptoms of choking while eating.

7. The miscellaneous group in table 1, in which the origin of the lesion was in the central nervous system, presented some interesting cases:

(*a*) A male with a syndrome indicating damage of the brain with medullary involvement had paralysis of the right vocal cord and associated paralysis of the right side of the soft palate and pharynx.

(*b*) A female with paralysis of the right vocal cord had a large acoustic neuroma on the right side. At operation the tumor was found to encroach on

the eighth, ninth, tenth and eleventh cranial nerves. This may be considered an immediate infranuclear lesion still within the cranial vault.

(*c*) A male presented the so-called syndrome of Schmidt, with involvement of the nucleus of the tenth and associated involvement of the nucleus of the eleventh nerve. The lesion was considered to be on a vascular basis in the left side of the medulla because of a Babinski sign on the right, paralysis of the left vocal cord and atrophy of the left sternocleidomastoid and trapezius muscles.

(*d*) A male was considered clinically to have a degenerative lesion of the brain stem and cerebellar pathways. He presented paralysis of the left recurrent laryngeal nerve.

(*e*) The last patient in this group also was found to have cerebroarteriosclerotic changes that involved the bulb as well as the cortex. The question was raised clinically whether this man had a pseudobulbar palsy. The left vocal cord was paralyzed and lying in the midline.

8. The miscellaneous group in the paretic series (table 2) includes 6 male and 1 female patients.

(*a*) The one female showed clinically an acute ascending myelitis with bulbar involvement. The left vocal cord was paretic.

(*b*) A male had been the victim of multiple cerebral vascular accidents and presented paresis of the right facial nerve and the right vocal cord.

(*c*) A male presented a bilateral degenerative cerebellar syndrome and bilateral abductor paresis of the vocal cords.

(*d*) A male showed signs of pontile hemorrhage after an injury to the head in an automobile accident. This was later followed by a syndrome indicating a lesion in the left half of the cerebellum. He had paresis of the left facial nerve and left vocal cord.

(*e*) A male presented clinical indications of multiple small cerebral thrombi. Bilateral abductor paresis of the vocal cords was noted.

(*f*) A male was suspected to have a brain tumor or to have had a vascular accident. He presented hemiparesis of the right side of the body and paresis of the right vocal cord. A ventriculogram was refused, and the patient was subsequently lost track of.

(*g*) The last case in this group was similar to (*f*). The patient had a cerebral hemorrhage on the right with hemiparesis of the right side of the body and paresis of the right vocal cord.

9. The case of paralysis of the vocal cord associated with poliomyelitis is self explanatory.

10. Although it is generally conceded that paralysis of the vocal cord cannot accompany lesions above the nucleus ambiguus, I believe that transient paresis of supranuclear origin is often missed clinically. The transient and often unrecognized paresis associated with syphilis and affecting various parts of the body is well known. Paresis of central origin is recognized only if the compensatory function of the other portions of the cortex is delayed. It was in this interval that paresis in the foregoing cases was noted. As will be shown later, the patient in case 8 *e* was reexamined at a later time and showed complete recovery.

11. Fifty-three per cent of the patients with central lesions were in the decade of 50 or above. Males were affected in comparison with females in the ratio of 2.3 to 1. In 4 cases of bilateral abductor paralysis of the vocal cords in the paralytic group the condition was associated with a bulbar lesion, while in 4 cases of bilateral paresis of the vocal cords it was associated with a supranuclear lesion. In the paralytic group, the left cord was affected in relation to the right in a ratio of approximately 2:1, and in the paretic group the right and the left cord were affected about equally. In the former group the cord was found in the median position approximately three times as often as in the cadaveric position.

CASES OF DYSFUNCTION DUE TO OPERATION ON THE THYROID GLAND

By far the largest group of cases was that in which the dysfunction of the vocal cord was associated with operative procedures on the thyroid gland. It is customary in the University Hospital for a patient to have a routine laryngeal examination preoperatively and a check-up examination postoperatively. The postoperative examination is usually within the first week following operation and before the patient is discharged. It is possible that instances of transient paralysis or paresis of the vocal cords that occur immediately postoperatively are missed. The importance of this routine procedure was emphasized elsewhere by Zinn,[8] New[9] and Matthews.[10]

The anatomy of the laryngeal nerves has been sufficiently described by Joll,[11] Piersol,[12] Dilworth,[13] Ziegelman[14] and Lemere.[15]

Ziegelman[14] showed nicely the possible points of injury to these nerves.

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8. Zinn, W. F.: Postoperative Bilateral Abductor Paralysis, Ann. Otol., Rhin. & Laryng. **44**:164-169 (March) 1935.

9. New, G. B.: The Larynx in Diseases of the Thyroid, Ann. Clin. Med. **1**: 262-265 (Jan.) 1923.

10. Matthews, J.: Observations on the Vocal Cords in One Thousand Cases of Goiter, J. A. M. A. **55**:826-828 (Sept. 3) 1910.

11. Joll, C. A.: Discussion on the Laryngeal Paralyses, Proc. Roy. Soc. Med. **28**:202-206 (Dec.) 1934.

12. Piersol, G. A.: Human Anatomy, Philadelphia, J. B. Lippincott Company, 1930, pp. 1270-1271.

13. Dilworth, T. F. M.: The Nerves of the Human Larynx, J. Anat. **56**: 48-52 (Oct.) 1921.

14. Ziegelman, E. F.: Laryngeal Nerves, Arch. Otolaryng. **18**:793-808 (Dec.) 1933.

15. Lemere, F.: Innervation of the Larynx, Arch. Otolaryng. **18**:413-424 (Oct.) 1933.

Injury to the recurrent nerves may be temporary or permanent. Judd, New and Mann [16] found by experiments on dogs that stretching had little effect on the function of the recurrent laryngeal nerves, that pinching of these nerves at the inferior pole of the gland or closer to the larynx caused paralysis but that recovery ensued and, finally, that tying or severing the nerve caused complete paralysis that was probably permanent. As New [9] pointed out, paralysis of the vocal cord may be due to hemorrhage and edema, and, if so, is temporary. Physicians who have followed patients who were operated on for thyroid disease are well aware that paralysis may not be immediate. Postoperative examination of the larynx may show nothing abnormal, yet the patient will return at a later date with paralysis of one or even of both vocal cords. The explanation is probably to be found in the formation of scar tissue and involvement of the recurrent nerves.

The available statistics on dysfunction of the vocal cords following thyroidectomy are scanty as to the comparative incidence in males and females, the comparative incidence of partial and complete paralysis, the cord affected and the position of the cord. Tables 1 and 2 illustrate these points. In 16 per cent, or 14, of the cases in the paralytic group, there was bilateral paralysis of the vocal cords. The cords assumed the median position in 9 of these cases and the cadaveric in 2, and the position of the cords was unspecified in 3. Bilateral abductor types of paralysis of the vocal cords following thyroidectomy must be of particular interest to the laryngologist because of the well known danger of partial or complete laryngeal obstruction. This danger is particularly acute in cases in which thyroidectomy has been performed on a patient who is not a good surgical risk and in cases in which the patient suffers from thyrotoxicosis.

THREE CASES OF UNUSUAL CAUSATION

Paralysis of the recurrent laryngeal nerve was found after total pneumonectomy on the left. Because of the rareness of this type of complication, a short outline of the case will be presented.

CASE 1.—D. V., a white man aged 55, was first seen in the Department of Thoracic Surgery of the University Hospital in February 1937, with symptoms suggesting neoplasm of the lung. Bronchoscopic examination and biopsy on February 23 showed medullary carcinoma grade IV of the left lung. A left total pneumonectomy was performed on March 8. The patient's postoperative course was stormy but not unusual. Two weeks after operation hoarseness developed rather suddenly. On the twenty-second postoperative day, a diagnosis of paralysis of the left recurrent nerve was made. The position of the affected cord was not stated.

16. Judd, E. S.; New, G. B., and Mann, C. F.: The Effect of Trauma on the Laryngeal Nerves, Ann. Surg. **67**:257-262 (March) 1918.

Although no member of the Department of Otolaryngology made a preoperative laryngeal examination, I believe this case may rightly be classed as one of postoperative laryngeal paralysis following left total pneumonectomy, since:

1. No mention of paralysis of the vocal cord was made by the thoracic surgeon who passed, first, the laryngoscope and then the bronchoscope.

2. There was rather sudden onset of hoarseness postoperatively (two weeks), with no history of preoperative laryngeal difficulty (I realize that it is shown earlier in this paper that 30 per cent of patients do not have symptoms with paralysis of the laryngeal nerve).

3. Preoperative neurologic examination showed nothing abnormal.

CASE 2.—A girl aged 6 weeks had an enlarged thymus. Meningocele and spina bifida occulta were also present. During irradiation laryngeal stridor developed. She was seen on two different occasions by a member of the Department of Otolaryngology, and on each examination the right vocal cord was stationary in the cadaveric position.

CASE 3.—A man aged 28, with congenital heart disease, entered the hospital with a first attack of cardiac congestive failure. Although there were no symptoms referable to the larynx, laryngeal examination showed left abductor paralysis, the cord lying in the midline.

OTHER CASES

Tables 1 and 2 show adequately the other etiologic factors in laryngeal paralysis and paresis. The factors listed are in agreement with the types of causes set forth by New and Childrey [5] and by Smith, Lambert and Wallace.[6] In no case could a "chill," as listed by the latter authors, be found as the cause of paralysis; likewise, no cases were encountered in which the condition was due to toxins.

CORD AFFECTED AND POSITION IN FOREGOING GROUPS OF CASES

The left vocal cord was involved in paralysis (table 1) more frequently than the right one, both in the group in which the paralysis followed operation on the thyroid gland and in the groups of other causation. Bilateral abductor paralysis occurred more frequently in the postoperative group than in the combined other groups of cases in which the cause of dysfunction was known. In the cases of paralysis following thyroidectomy the median and the cadaveric positions of the cord occurred about equally, while in other groups the median position occurred in comparison to the cadaveric in the ratio of 3.6 to 1.

The left vocal cord was found to be more frequently affected also in the parietic group (table 2); this was especially true in the cases of postoperative paresis. Bilateral paresis was most frequently encountered in those cases in which there were lesions of the central nervous system.

TABLE 3.—*Follow-Up on Cases of Paralysis and Paresis Associated with Thyroidectomy*

Sex		Right Cord			Left Cord			Bilateral						
Male	Female	Median	Cadaveric	Unspecified	Median	Cadaveric	Unspecified	Median	Unspecified	No Return	Partial	Complete	Total	
							Paralysis							
	2+	2+	..	..	..	..	..	..	..	2+	..	..	2	
	4+	..	4+	..	..	..	..	..	..	4+	..	..	4	
+	..	..	..	+	..	..	..	..	..	+	..	..	1	Right 18
+	..	..	..	+	..	..	..	..	..	..	..	+	1	
	8+	..	..	8+	..	..	..	..	..	8+	..	..	8	
	2+	..	..	2+	..	..	..	..	..	..	..	2+	2	
	+	..	..	..	+	..	..	..	..	+	..	..	1	
	+	..	..	..	+	..	..	..	..	..	+	..	1	
	+	..	..	..	+	..	..	..	..	..	..	+	1	
+	..	..	..	..	..	+	..	..	..	+	..	..	1	Left 15
	2+	..	..	..	..	2+	..	..	..	2+	..	..	2	
	+	..	..	..	..	+	..	..	..	..	..	+	1	
	7+	..	..	..	..	..	7+	..	..	7+	..	..	7	
	+	..	..	..	..	..	+	..	..	..	..	+	1	
+	..	..	..	..	..	..	..	+	..	+	..	..	1	
+	..	..	..	..	..	..	..	+	..	..	..	+	1	
	2+	..	..	..	..	..	..	2+	..	2+	..	..	2	Bilateral 8
	+	..	..	..	..	..	..	+	..	..	+	..	1	
	2+	..	..	..	..	..	..	2+	..	..	..	2+	2	
	+	..	..	..	..	..	..	..	+	..	..	+	1	
Total 5	36	2	4	12	3	4	8	7	1	29	2	10	41	
							Paresis							
	+		+			..			..	..	..	+	1	Right 1
	+		..			+			..	..	..	+	1	
	4+		..			4+			..	4+	..	..	4	Left 7
	2+		..			2+			..	..	..	2+	2	
+	..		..			..			+	..	..	+	1	Bilateral 2
	+		..			..			+	..	+	..	1	
Total 1	9		1			7			2	4	1	5	10	

FOLLOW-UP EXAMINATIONS

Postoperative Cases.—In 1910 Matthews [10] stated: "No patient with total paralysis, observed either before or after operation [referring to thyroidectomy], was seen to have recovered, though, through lack of opportunity for reexamination after the lapse of considerable time, such recovery might have escaped notice."

Judd [17] stated that "the disturbance which sometimes follows immediately after the operation is temporary, and normal function will be restored in from a few days to a few weeks."

Feuz (quoted by Joll [11]) reported that in approximately 50 per cent of the cases of complete and incomplete recurrent nerve paralysis in the Lausanne series (250 cases) the condition disappeared within six to twelve months.

New and Childrey [5] stated that in 10 of 39 cases followed after thyroidectomy there was complete or partial recovery.

In this series, 41 cases (table 3) of total paralysis of the vocal cords occurring after thyroidectomy were followed postoperatively. In 10, or 24 per cent, there was complete recovery of function. In the 3 cases of paralysis of the right vocal cord in which there was complete return of function, the position of the cord at the original examination was not stated. Of those in which the left vocal cord was affected, with complete recovery the cord was in a median position in 1 and in the cadaveric position in 1; the position of the cord in the third was not specified. In the 4 cases of bilateral paralysis in which there was recovery the cords were all in the median position at the original postoperative examination.

The time necessary for recovery is important, especially if the patient has symptoms referable to the dysfunction of the vocal cord. In 5, or 50 per cent, of the 10 cases in which there was recovery, this occurred within one month; in 4 more cases there was recovery at the end of six months. Thus, 90 per cent of the patients who recovered did so within six months after operation. In the last case of the 10, there was recovery at the end of three years—when during this three year interval recovery took place cannot be stated.

In 5 cases, or 50 per cent, of the 10 in the paretic group which were followed postoperatively, there was recovery by the end of two months.

If both groups are combined, in 14 cases, or 27 per cent of the 51 followed, there was recovery at six months.

Cases Other Than Postoperative.—Smith, Lambert and Wallace [6] followed 20 cases of paralysis of the laryngeal nerve and found that in 5 there was complete return of function. In 4 of the 5 no cause for the paralysis had been discovered originally.

17. Judd, E. S.: Laryngeal Function in Thyroid Cases, Ann. Surg. **73**:321-327 (March) 1921.

TABLE 4.—*Follow-Up on Cases of Paralysis and Paresis Not Associated with Thyroidectomy*

Cause	Sex		Right Cord			Left Cord			Bilateral				
	Male	Female	Median	Cadaveric	Unspecified	Median	Cadaveric	Unspecified	Unspecified	No Return	Partial Return	Complete Return	Total
Paralysis													
Unknown	+		..	+	..	..	..	..	..	+	..	..	1
Unknown	+		..	..	+	..	..	..	..	+	..	..	1
Unknown	2+		..	..	..	2+	..	..	..	2+	..	..	2
Unknown		+	..	..	..	..	+	..	..	..	..	+	1
Unknown	+		..	..	..	..	..	+	..	..	+	..	1
Unknown	+		..	..	..	+	..	..	..	+	..	..	1
Unknown	+		..	..	..	..	..	+	..	+	..	..	1
Unknown	+		..	..	..	..	..	..	+	+	..	..	1
Poliocencephalitis		+	..	..	..	..	..	+	..	+	..	..	1
Syphilis of the central nervous system	+		..	..	..	..	..	..	+	+	..	..	1
Tumor of the hypopharynx and esophagus	+		..	..	..	..	..	..	+	+	..	..	1
Benign goiter		+	+	..	..	..	..	..	..	+	..	..	1
Malignant goiter	+		..	+	..	..	..	..	..	+	..	..	1
Malignant goiter		+	..	..	..	+	..	..	..	+	..	..	1
Apical fibrotic tuberculosis		2+	..	..	2+	..	..	..	..	2+	..	..	2
Apical fibrotic tuberculosis	+		..	..	..	..	..	+	..	+	..	..	1
Inflammation of the mediastinal glands		+	..	..	..	..	+	..	..	+	..	..	1
Enlarged thymus		+	..	+	..	..	..	..	..	+	..	..	1
Total	12	8	1	3	3	4	2	4	3	18	1	1	20
Paresis													
Unknown		+		..			+		..	..	..	+	1
Cerebral hemorrhage	+			+			..		..	..	..	+	1
Nongoitrous tumor of the neck	+			+			..		..	+	..	..	1
Mitral stenosis	+			..			+		..	..	..	+	1
Total	3	1		2			2		..	1	..	3	4

New and Childrey [8] found that 6 of 26 patients with paralysis of varied causation recovered partially or completely.

Table 4 shows data on 20 patients with total paralysis due to varied causes, whose cases were followed. The check-up examinations were made after intervals varying from one month to six and one-half years. In 1 case, or 5 per cent, in which the paralysis was of unknown cause, there was complete return of function, while in another in which the cause was unknown, there was partial return of function.

In 3, or 75 per cent, of the 4 cases in the paretic group, there was recovery. Of particular interest is the case of cerebral hemorrhage in which recovery was shown on reexamination. The interval before reexamination was three years, but during this time the patient's general condition improved, with coincident return of the function of the vocal cord.

CONCLUSIONS

1. Paralysis of the recurrent nerve occurs later in life in males than in females, if paralysis associated with operation on the thyroid gland is disregarded.

2. Thirty per cent of patients with paralysis of the vocal cord have no laryngeal symptoms. Fifty per cent have no symptoms with paretic involvement of the vocal cords.

3. Transient paretic conditions of the vocal cords associated with central cerebral lesions exist; they may be unilateral or bilateral and are probably missed clinically for the most part.

4. Postoperative paralysis of the recurrent laryngeal nerves associated with thyroidectomy occurs in females and in males in a ratio of approximately 7.4 to 1. The left nerve is slightly more often involved. The median and cadaveric positions of the cords occur with equal frequency. Bilateral impairment of the vocal cords occurs about once in every 6 cases of postoperative paralysis associated with thyroidectomy, and in this group the median position of the vocal cords is most often encountered.

5. Paralysis of the laryngeal nerve may occur after one stage total pneumonectomy.

6. If one disregards cases in which postoperative paralysis is associated with thyroidectomy, the left vocal cord is more often affected by paralysis than the right. The median position of the cords is more often encountered than the cadaveric.

7. Prognosis for recovery of the vocal cords is approximately 25 per cent in cases of postoperative paralysis associated with thyroidectomy and 50 per cent in cases of paresis. Recovery, if it takes place, usually occurs within six months.

8. Prognosis for recovery from paralysis other than the postoperative type is poor (5 per cent); in the paretic group the prognosis is better.

CROUP

PRELIMINARY REPORT ON ONE YEAR'S INVESTIGATION OF TWO HUNDRED AND TWENTY-SIX CASES

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The term "croup" is used to designate a state of obstruction to the entrance of air into the lungs as the result of an inflammatory process either in the larynx alone or extending to the lower respiratory tract. It is characterized by inspiratory stridor, hoarseness or aphonia and cyanosis or pallor, accompanied by suprasternal, epigastric and intercostal retractions.

There are two main types, diphtheritic and nondiphtheritic.

Formerly most attention was focused on the diphtheritic type, since that type was the principal cause of obstruction. Before extensive use of toxin-antitoxin, 60 per cent of the cases of croup were of the diphtheritic type, but in the past year the incidence of diphtheritic croup fell to only 7 per cent. Of 450 cases of croup encountered at the Kingston Avenue Hospital in 1929, 316 were of the diphtheritic and 134 of the nondiphtheritic type. In the year beginning April 1, 1939 and ending March 31, 1940, of 226 cases, 16 were of the diphtheritic and 210 of the nondiphtheritic type.

In spite of the tremendous decrease in the incidence of diphtheritic croup, the number of cases of nondiphtheritic croup has remained about the same. The investigation reported here concerned itself primarily with nondiphtheritic croup, which was more prevalent at the Kingston Avenue Hospital in 1939 than in any previous year. Nondiphtheritic croup has been referred to as "acute catarrhal laryngitis," "pseudomembranous croup," "fibrinous croup," "acute laryngotracheobronchitis," "acute fulminating laryngotracheobronchitis," "laryngitis subglottica," "influenzal laryngitis," "phlegmonous laryngitis," "acute streptococcic laryngitis" and by other names.

Read before the Brooklyn Academy of Pediatrics, March 27, 1940.

From the Diphtheria Service, Kingston Avenue Hospital, Brooklyn, New York City Department of Hospitals.

The multiplicity of names indicating croup is confusing and may imply arbitrary interpretation of the lesion or its underlying pathologic conditions by different observers. Failure to standardize the nomenclature constitutes a challenge to those responsible for the care of patients with this disease. This challenge may be considered answered only after a proper classification of croup has been accepted.

A careful analysis of old hospital records revealed insufficient information for dependable evaluation of various types of croup. We therefore deemed it timely to create a new chart, which would provide the desired detailed information in simplified form. This chart is so arranged as to permit the recording of daily observations by the attending physician or intern. It is offered for consideration in the recording of data concerning patients with croup.

A practical classification should automatically yield a clue to the appropriate management as well as to the immediate prognosis in each case of croup.

TABLE 1.—*Chart for Patient with Croup*

Name	Age	Date of Admission	Hospital No.	History	Physical Findings and Roentgenograms	Diagnosis on Admission	Instrumentation and Indications	Findings on Instrumentation	Immediate Result of Instrumentation	Bacteriologic Findings	Postoperative Treatment	Result

As the result of careful study of the data obtained on the new charts, we submit what we consider a comprehensive classification of cases of croup. We do so with the realization that any classification will be subject to criticism of one kind or another.

I. Diphtheritic croup
   1. Diphtheritic obstructive laryngitis
   2. Diphtheritic obstructive laryngotracheitis
   3. Diphtheritic obstructive laryngotracheobronchitis

II. Nondiphtheritic croup
   1. Acute catarrhal laryngotracheitis
   2. Supraglottic edematous obstructive laryngitis
   3. Subglottic obstructive laryngitis and tracheitis
      (*a*) Subglottic ***exudative*** (or inspissated) obstructive laryngitis and tracheitis
      (*b*) Subglottic ***edematous*** obstructive laryngitis and tracheitis
   4. Acute obstructive laryngotracheobronchitis
   5. Unclassified (noninfectious)
      (*a*) Due to allergy
      (*b*) Due to foreign body
      (*c*) Due to chemicals and other irritative agents

DIPHTHERITIC OBSTRUCTIVE LARYNGITIS

Diphtheritic obstructive laryngitis is a benign form of croup characterized by only moderate laryngeal stridor, croupy cough, hoarseness or aphonia and mild suprasternal and epigastric retractions. The symptoms are usually relieved by administration of diphtheria antitoxin, 20,000 units given intramuscularly and 10,000 units given intravenously. Laryngoscopic examination reveals a nonedematous larynx with scattered patches of membrane above and on the vocal cords, not sufficient as a rule to give rise to alarming symptoms.

DIPHTHERITIC OBSTRUCTIVE LARYNGOTRACHEITIS

In diphtheritic obstructive laryngotracheitis, a dirty grayish membrane is present both in the larynx and in the trachea, where it is usually accompanied by a mucopurulent exudate responsible for hoarseness or aphonia; other manifestations are suprasternal and epigastric

Fig. 1.—Suction cannula (Kingston Avenue Hospital model) with standard adapter. The length is 45 cm. and the diameter of the lumen is 3.5 mm.

retractions and cyanosis or extreme pallor. The chemotherapy consists in the administration of 20,000 units of diphtheria antitoxin intramuscularly and 10,000 units intravenously. The diphtheritic membrane is removed by laryngoscopic suction and the use of long cotton-wound applicators moistened with a saline solution; these are passed between the vocal cords into the trachea. Usually this quickly relieves the retractions and reduces cyanosis. Whenever retractions and cyanosis recur, laryngoscopic suction is repeated. The prognosis is generally good despite frequent repetition of direct suction.

DIPHTHERITIC OBSTRUCTIVE LARYNGOTRACHEOBRONCHITIS

Patients with diphtheritic obstructive laryngotracheobronchitis are extremely ill. They present marked circumoral pallor, suprasternal and epigastric retractions and restlessness for want of air. If these symptoms are not relieved, a state of exhaustion may ensue, which must not be mistaken for so-called toxic diphtheria.

Laryngoscopic inspection reveals a membrane in the larynx, extending between the vocal cords into the trachea. Bronchoscopic examination discloses extension of the membrane into the bronchi, narrowing or occluding their lumens. The chemotherapy consists in the intramuscular injection of 20,000 units of diphtheria antitoxin and the intravenous injection of 10,000 units. Suction through the bronchoscope may not relieve the patient's symptoms, and tracheotomy becomes imperative. Postoperative care consists of the use of the bronchoscope through the tracheotomy wound, to facilitate visualization and for suctioning of the bronchi. If suction fails, the obstructing membrane may be removed with a forceps. In some instances the membrane is seen plugging the bronchioles. Direct suction removes the plugs and restores aeration of previously atelectatic areas.

Severe recurrent attacks of respiratory obstruction, with a rapid increase in the breath rate, cyanosis, retraction and restlessness, require that the nurse aspirate such membrane or secretions as she can through the tracheotomy cannula. If this fails to relieve the distress, the resident physician removes the tracheotomy tube and inserts a smooth hollow metal suction tube through the tracheotomy wound. The suction tube is passed down to the carina or beyond into the bronchi.

If this procedure does not relieve the cyanosis and retractions, a bronchoscope is passed through the tracheotomy wound into the bronchi on each side, where under direct observation proper suction is applied; this usually suffices to relieve the cyanosis and retractions.

Manipulations through the tracheotomy wound, although performed without anesthesia, are usually well tolerated; in fact, patients respond to the removal of obstructing membrane in the lower bronchial tree by falling into a sound sleep, with a sudden drop in the respiratory rate, or by sitting up after the procedure, taking an interest in their surroundings and asking for water or food. The change may be dramatic. An hour or more later, however, signs of urgent distress may reappear. Treatment with the bronchoscope should be repeated as often as such attacks recur. Anoxemia from endobronchial obstruction requires prompt and energetic treatment. This clinical entity must not be confused with so-called toxic diphtheria, lest the patient be deprived of the lone chance of recovery.

J. C., aged 2½ years, required tracheotomy and numerous suctions through a cannula by a special nurse; 22 suctions through the tracheotomy wound and 5 bronchoscopic suctions by the same route were performed before recovery was assured. On several occasions, before the recurring membranes were removed, this patient appeared as though about to die.

Obstructive laryngotracheobronchial diphtheria formerly had an appalling mortality rate, but in the past year we had 2 patients with such a condition, both of whom survived after a stormy session.

Complicating pneumonias were treated by the attending pediatricians in the usual manner.

When there was a tendency to dry crusting, progress was favorably influenced by the use of a steam tent.

ACUTE CATARRHAL LARYNGOTRACHEITIS

Patients suffering from acute catarrhal laryngitis and tracheitis have mild suprasternal and epigastric retractions and little or no cyanosis and appear to be only moderately ill. They have mild brassy cough and slight hoarseness. Laryngoscopic procedure is unnecessary in most cases. If, however, there is any doubt as to the type of lesion to be dealt with, direct laryngoscopic inspection should be done. It will usually reveal a deeply injected larynx with some mucoid secretion on the vocal cords and extending into the subglottic area. This secretion is easily aspirated and leaves a reddened mucosa in the subglottic area and upper part of the trachea. The cords are observed to move freely.

TABLE 2.—*Cases of Diphtheritic Croup*

	Cases	Deaths	Cures
Diphtheritic obstructive laryngitis	5	0	5
Diphtheritic obstructive laryngotracheitis	9	1	8
Diphtheritic obstructive laryngotracheobronchitis	2	0	2
Totals	16	1 (6%)	15 (94%)

Placing the patient in a steam tent is usually sufficient to effect a cure. The prognosis is good. Of 154 patients, all were cured but 1, whose death was due to pneumonia and a complicating cardiac lesion.

SUPRAGLOTTIC EDEMATOUS OBSTRUCTIVE LARYNGITIS

Supraglottic edematous obstructive laryngitis causes marked suprasternal and epigastric retractions, loud, noisy inspiratory stridor, a peculiar "hot potato" voice, hoarseness, brassy cough, marked cyanosis or pallor and usually a temperature of 103 F. or more. The patient assumes a characteristic sitting position with chin forward and mouth wide open. Placing the patient in the recumbent position immediately increases cyanosis and stridor. Direct laryngoscopic inspection discloses a red swelling of the epiglottis, aryepiglottic, folds, arytenoids and false cords. In some instances not all of these structures are involved. The most common sites are the epiglottis and arytenoids. Once recognized, this condition is an indication for immediate tracheotomy, preferably with the bronchoscope in situ. Intubation is contraindicated, because the swollen epiglottis, aryepiglottic folds and arytenoids tend to overlap the upper opening of the intubation tube and completely block the airway. The

fact that the mucosa is intact suggests that it does not play an active part in the spread of the infection in this type of lesion. Included in the series reported here are 7 cases of supraglottic edematous obstructive laryngitis. All of the patients were cured, 6 by tracheotomy and 1, with a mild condition, by inhalation of steam. A striking case was that of a child who died within four hours after the onset of marked retractions and within a few minutes of admission to a hospital, before anything could be done for him. Immediate postmortem endoscopic examination revealed marked purplish edematous swelling of all the supraglottic structures without any subglottic edema. Gross dissection of the larynx post mortem likewise confirmed the absence of subglottic edema, although the intense swelling of the supraglottic structures was responsible for obstruction to the glottic airway. It is our contention that patients with supraglottic edematous obstructive laryngitis rarely, if ever, show coexisting subglottic edema. Consequently, we believe croup of this type to be a localized, self-limited inflammatory process (figs. 2, 3 and 4). Relief of the extreme anoxemia by timely tracheotomy will effect a cure in practically all such cases. Croup of this type is most frequently encountered in children of 3 years or more.

SUBGLOTTIC OBSTRUCTIVE LARYNGITIS AND TRACHEITIS

Exudative (or Inspissated) Type.—The exudative (or inspissated) type of subglottic obstructive laryngitis and tracheitis is characterized by intense suprasternal and epigastric retractions, cyanosis, hoarseness and brassy cough. Laryngoscopic inspection usually reveals that the vocal cords are immobile, nearly in the midline and only slightly separated by moist exudate and/or dry crusts, which form in the subglottic area. This exudate and crust have a greenish, grayish or blackish appearance, depending on the aggregate of dirt particles adherent to the surface. Laryngoscopic suction of this material or its removal with a forceps permits the cords to move freely again, disclosing a congested, nonedematous mucosa in the subglottic space. Immediate relief of cyanosis and respiratory distress follows, and patients either fall into a sound sleep or sit up and ask for fluids or food. Occasionally two or more laryngoscopic suctionings become necessary because of the recurrence of alarming signs. The prognosis is good. Seventeen patients with a condition such as this have been treated without a fatality. We have observed 2 in whom croup beginning as the subglottic exudative obstructive type gradually progressed to the subglottic edematous obstructive stage, which required more drastic treatment after laryngoscopic suctioning.

It is probable that in the past many successful intubations for the relief of nondiphtheritic croup were performed on patients with croup of this type. In cases such as this, laryngoscopic suctioning affords a safer, more conservative and consistently successful method of treatment. Acute catarrhal laryngitis and tracheitis may advance to the subglottic exudative obstructive stage.

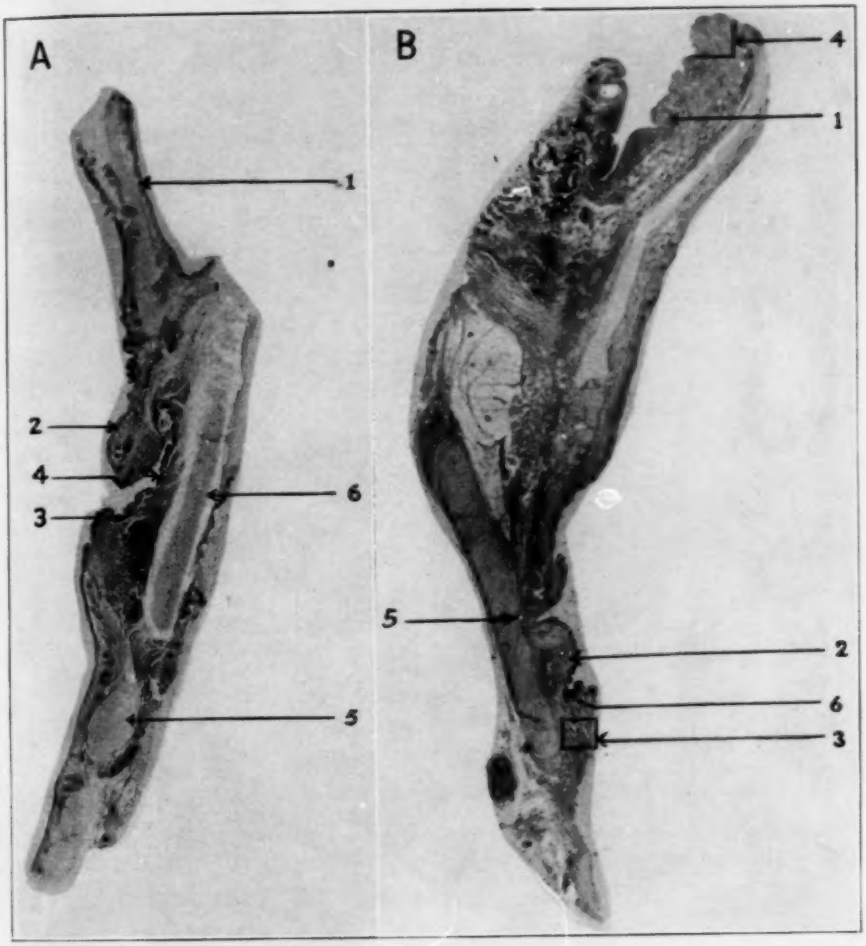

Fig. 2.—*A*, longitudinal section of a normal larynx (× 1.9). *1* indicates the tongue surface of the epiglottis; *2*, the false vocal cord; *3*, the true vocal cord; *4*, the ventricle; *5*, the cricoid ring, and *6*, the thyroid cartilage. *B*, longitudinal section of a larynx showing supraglottic edematous obstructive laryngitis with swelling and inflammatory involvement of the supraglottic region, especially of the tongue surface of the epiglottis. *1* indicates edema of the tongue surface of the epiglottis; *2*, the vocal cord, and *3*, the normal subglottic area (*4*, *5* and *6* will be referred to in the legends to figures 3 and 4).

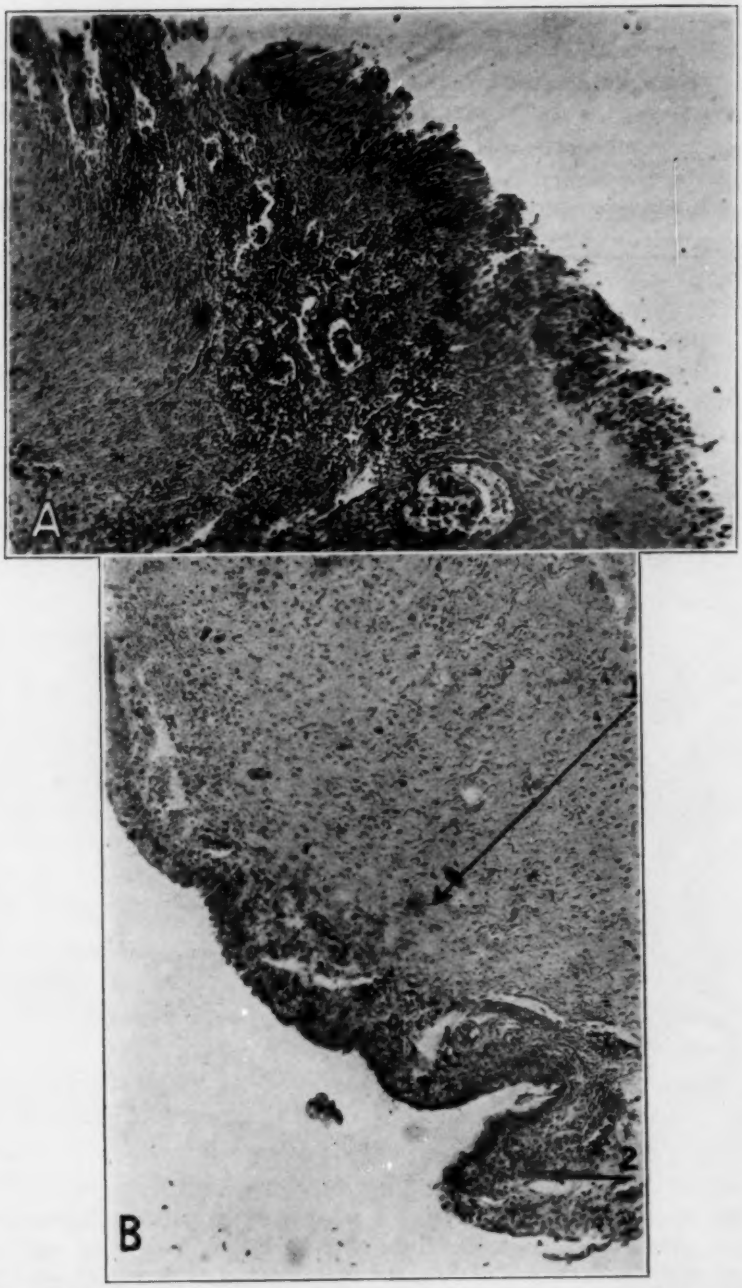

Fig. 3.—*A*, tip of the epiglottis (*4* in fig. 2 *B*), disclosing hemorrhage, edema, necrosis and extensive exudate, consisting of polymorphonuclear leukocytes and round cells (× 50). *B*, junction of the false and the true vocal cord (*5* in fig. 2 *B*), showing slight edema and round and occasional polymorphonuclear cells in the region of the false vocal cord and practically no involvement of the true cord. *1* indicates the false cord and *2* the true cord.

Edematous Type.—The edematous type of subglottic obstructive laryngitis and tracheitis causes hoarseness, brassy cough, cyanosis or pallor, marked suprasternal and epigastric retractions and rapid pulse. Direct laryngoscopic inspection reveals the supraglottic structures to be practically normal and the vocal cords fixed approximately in the midline. Suctioning under vision fails to influence the relative position of the cords or to increase their mobility. This is accounted for by the presence of subglottic edema and infiltration (figs. 5 and 6). The sub-

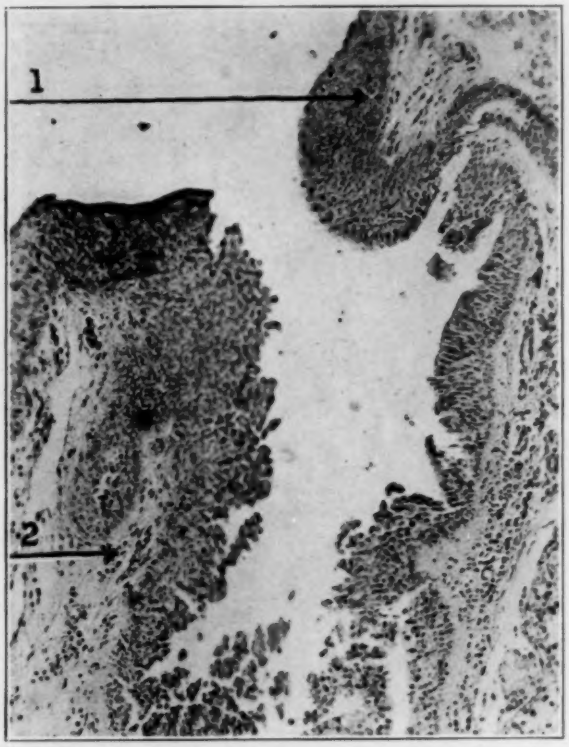

Fig. 4.—Slight round cell infiltration in the mucous membrane of the subglottic area, which is otherwise normal (*6* in fig. 2 *B*). *1* indicates the true cord and *2* the subglottic area.

glottic edematous swelling prevents the entrance of air into the lungs in two ways: (1) by its physical presence below the cords and (2) by interference with the motion of the cords, which cannot abduct during inspiration. Having decided on the nature of the disease at hand, the operator is faced with a choice of two procedures, depending on the extent of the involvement.

If one can be reasonably certain that the subglottic obstructive edema is confined to an area immediately below the cords and the first few

rings of the trachea, there may be justification for intubation by the direct method. This alone does not guarantee permanent relief, because thick exudate coming from the trachea and bronchi may block the lumen

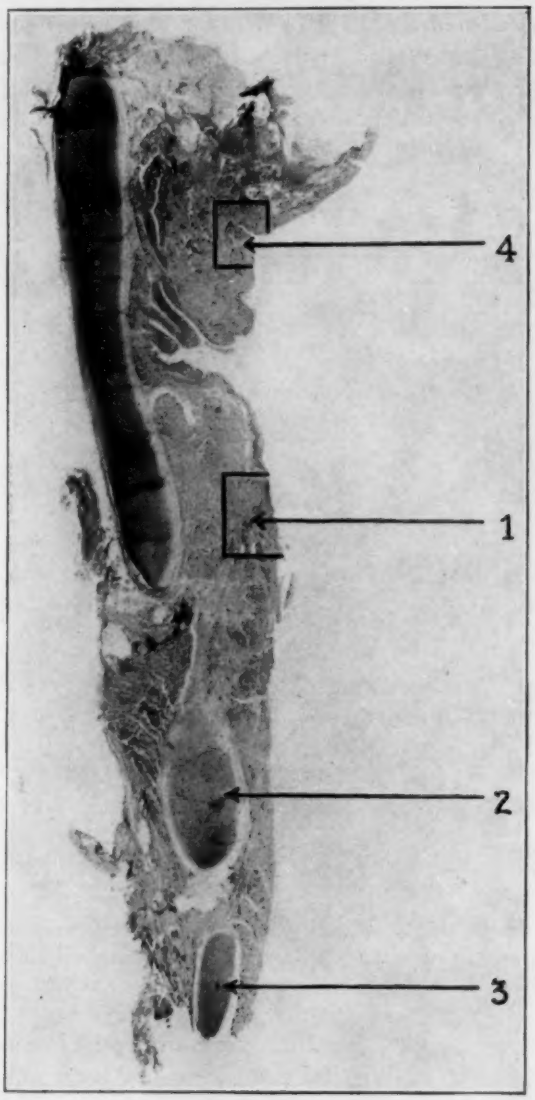

Fig. 5.—Longitudinal section (× 1.9) of the larynx in the case of subglottic edematous obstructive laryngitis. The supraglottic region is normal. *1* indicates extensive subglottic edema; *2*, the cricoid ring; *3*, the first tracheal ring, and *4*, the junction of the epiglottis and the false cord (normal).

of the intubation tube without warning and cause a recurrence of the distressing signs which first prompted intervention. The subglottic

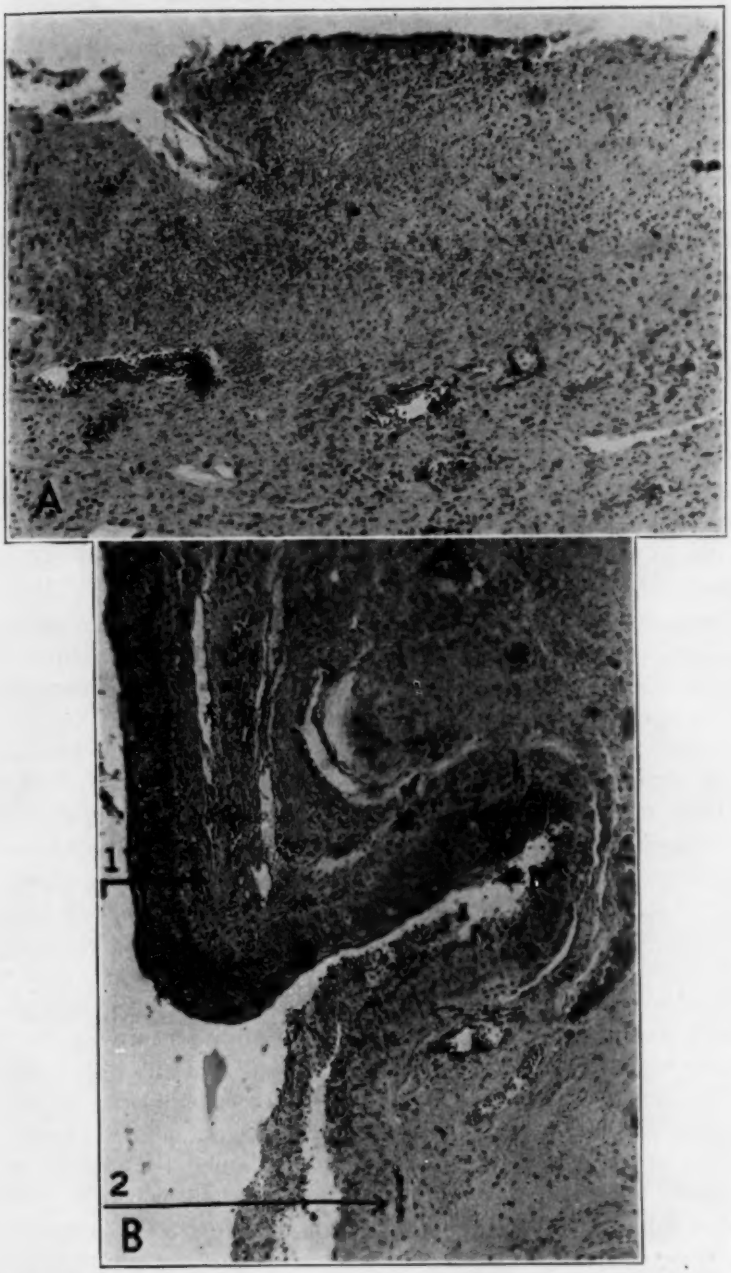

Fig. 6.—*A*, section from the subglottic area (*1* in fig. 5), showing extensive mucosal necrosis with fibrinous deposition and exudate consisting of polymorphonuclear leukocytes and occasional red blood cells (× 50). *B*, junction of the epiglottis and the false vocal cord (*4* in fig. 5) (× 50). The mucosa shows slight congestion. Otherwise it is normal. *1* indicates the epiglottis and *2* the false cord.

laryngeal and tracheal edema may spread down and cover the lower end of the tube, again provoking retractions, cyanosis and other indications of involvement. Not infrequently, autoextubation may occur, with return of the original symptoms, after which reintubation may not give relief. Repeated autoextubation causes anxiety to the patient and to the physician, lest the occurrence go unnoticed. A final decision must be made promptly regarding the alternate procedure (tracheotomy).

Laryngeal stenosis may result from prolonged intubation or frequent reintubation. The insertion of oversized tubes and their forceful removal may be responsible for extensive damage to tissues and consequent atresia. Lesser evils from the use of intubation tubes are ulcerations of the larynx and trachea, pretracheal abscess, webs of the larynx and fixation of the cords.

If one decides on intubation, the procedure should be carried out under direct vision, so that any pathologic condition in the larynx can be visualized at the same time. In the interest of good laryngeal function the indirect method of intubation is discouraged and the non-cough-up tube condemned.

From our limited experience with direct intubation in subglottic edematous obstructive laryngitis and tracheitis, we are compelled to recognize the overwhelming advantage of tracheotomy and recommend it as the operation of choice in cases of this class.

Patients with croup of this type associated with frank pneumonia may be encountered. They are subject to the same logical consideration, their treatment being dependent primarily on the interpretation of the laryngeal picture. Besides the signs and symptoms of pneumonia, they present marked suprasternal and epigastric retractions, pathognomonic of laryngeal obstruction. Pneumonia is no contraindication to procedures directed to the relief of urgent respiratory embarrassment, any more than it would be to the removal of an obstructing foreign body.

Patients with pneumonia in the terminal stage may at first glance resemble those suffering from croup. Clinically they present dyspnea, cyanosis, high fever and toxemia. They appear critically ill and breathe very rapidly. They have shallow xiphoid retractions, which may include the lower few ribs. Close observation, however, fails to reveal any alteration of voice or the suprasternal or deep xiphoid retractions which are present in cases of true croup. Such patients are not to be admitted to the croup wards, and they should not be subjected to instrumentation for the purpose of differential diagnosis. Postmortem examination invariably reveals extensive involvement of the lungs and absence of obstructive laryngeal and tracheal lesions.

The edematous type of subglottic obstructive laryngitis and tracheitis occurs most frequently in children under 2 years and is rarely encountered in older children. Infants normally have a small subglottic space

and a narrow tracheal lumen. Because of these anatomic facts, relatively little edema may diminish or close the airway to the lungs. It is worthy of mention that in cases of subglottic edematous obstructive laryngitis the infection seems to be limited to the subglottic space and the supraglottic structures remain uniformly free of involvement.

The prognosis depends on the choice of procedure. Experience has taught us to rely less and less on intubation. We are convinced by the striking difference in mortality rate that tracheotomized children have a far better chance. Steam and the oxygen tent are indispensable adjuncts to treatment. Concurrent pneumonia is a serious complication and alters the outlook considerably.

The edematous type of obstructive subglottic laryngitis and tracheitis should receive more attention in the literature than it has in the past. In cases of this type we confidently anticipate an appreciable reduction of the present high mortality rate. Better understanding of the nature of the lesion and logical management of the patient will bring this about.

TABLE 3.—*Instrumentation in Cases of Subglottic Edematous Obstructive Laryngitis and Tracheitis*

Procedure	Cases	Cures	Deaths
Intubation	6	2	4 (66%)
Intubation and tracheotomy	4	2	2 (50%)
Tracheotomy	5	4	1 (20%)
Suction (mild case)	1	1	0
Total	16	9	7 (45%)

ACUTE OBSTRUCTIVE LARYNGOTRACHEOBRONCHITIS

Patients with acute obstructive laryngotracheobronchitis are gravely ill, and aside from a toxic appearance they present a pale cyanosis, pronounced suprasternal and epigastric retractions, high mounting temperature, rapid pulse and increased respirations. There frequently are atelectasis and associated bronchopneumonia. This condition is usually seen in children under 2 years.

Inasmuch as available literature on the subject conveys conflicting views about this malady, a description of acute obstructive laryngotracheobronchitis may help clear up current confusion. Direct laryngoscopic inspection shows the cords fixed approximately in the midline. Obstructive edema is present below the cords and extends down into the trachea and primary and secondary bronchi. The trachea, bronchi and bronchioles contain mucopurulent exudate or a gummy material which prevents the inspiration of sufficient air. Areas of pulmonary atelectasis result from obstruction of the smaller bronchioles by the edema and exudate. Obstructive emphysema generally accompanies the atelectasis, and when retraction is severe and prolonged, interstitial emphysema with pneumothorax may develop.

The term "acute *obstructive* laryngotracheobronchitis" is used in preference to "acute laryngotracheobronchitis" or "acute fulminating laryngotracheobronchitis" because the element of obstruction is the predominant feature.

Acute hemorrhagic necrosing tracheobronchitis associated with acute hemorrhagic pneumonitis (so-called influenzal pneumonitis) but without subglottic edema should not be included. Pneumonia with associated mild catarrhal inflammation of the laryngotracheobronchial tree is excluded. It is important to note that several cases of subglottic edematous laryngitis and tracheitis have apparently been considered and reported by various observers as cases of obstructive laryngotracheobronchitis.

Recently, obstructive laryngotracheobronchitis has enjoyed prominence in the literature to the exclusion of most other varieties of croup. Careful analysis of the related pathologic conditions, symptoms and clinical data reported leaves considerable doubt as to the accuracy of diagnosis in some cases. Fortunately, cases of genuine acute obstructive

TABLE 4.—*Instrumentation in Cases of Acute Obstructive Laryngotracheobronchitis*

Procedure	Cases	Deaths	Cures
Intubation	2	2	0
Intubation and tracheotomy	2	2	0
Tracheotomy	4	3	1
Total	8	7 (87.5%)	1 (12.5%)

laryngotracheobronchitis are relatively rare (constituting about 3.5 per cent of our series). Under most favorable circumstances the mortality rate approximates 60 per cent. Most of our patients died within twenty-four hours after admission to the hospital, their temperatures having risen rapidly to 106 and 107 F.

By way of repetition: *Classic* acute obstructive laryngotracheobronchitis as seen by the laryngologist reveals congestion of the larynx and fixation of the true vocal cords approximately in the midline, with exudate extending up from below. Thorough suction of this exudate does not influence the mobility of the cords. Endoscopic examination reveals the trachea and bronchi filled with exudate or gummy material, on removal of which the underlying mucosa is found to be edematous and ulcerated. The tracheal lumen is so narrowed at times as to make passage of the bronchoscope difficult (or impossible). If the operator is fortunate enough to reach the tracheal bifurcation, he may find further progress impeded by edema in the main bronchi. No doubt remains as to the true nature of such a condition. Immediate tracheotomy must be performed and the bronchoscope passed through the wound for more efficient inspection and treatment of the lower part of the trachea and

bronchial trees. Management after tracheotomy consists of repeated suctioning through the tracheotomy cannula by the nurse. If cyanosis, pallor, retractions and other distressing signs continue, the tracheotomy tube is removed by the resident physician, and aspiration of the thick secretions with an especially designed suction tube is carried out through the tracheal opening. Should this prove ineffectual, the laryngologist then passes a bronchoscope through the wound to the carina or beyond and aspirates the obstructive secretions. These procedures may have to be repeated as frequently as plugging occurs. No other class of patients with croup require such constant attention.

TABLE 5.—*Incidence of Croup of Various Types Among the Patients Admitted to the Croup Service of the Kingston Avenue Hospital from April 1, 1939 to March 31, 1940*

Type of Croup	April	May	June	July	August	September	October	November	December	January	February	March	Cures	Deaths	Total
Diphtheritic obstructive laryngitis	1	..	1	..	..	1	..	..	..	..	2	..	5	0	5
Diphtheritic obstructive laryngotracheitis	2	1	..	2	..	1	..	..	..	..	1	2	8	1	9
Diphtheritic obstructive laryngotracheobronchitis	1	..	..	..	1	..	..	..	..	..	..	..	2	0	2
Acute catarrhal laryngotracheitis	5	3	..	3	8	15	17	17	20	28	26	12	153	1	154
Supraglottic edematous obstructive laryngitis	2	..	..	1	..	..	..	..	1	2	..	1	6	1	7
Subglottic exudative obstructive laryngitis and tracheitis	1	..	..	1	..	..	1	3	3	3	4	1	17	0	17
Subglottic edematous obstructive laryngitis and tracheitis	1	1	..	..	..	..	1	2	3	6	2	..	8	8	16
Acute obstructive laryngotracheobronchitis	..	..	..	..	..	2	1	..	1	..	2	2	1	7	8
Unclassified (allergic)	..	..	..	..	1	..	1	1	2	3	..	..	8	0	8
Total	13	5	1	7	10	19	21	23	30	42	37	18	208	18	226

Diphtheritic obstructive laryngotracheobronchitis and, occasionally, subglottic edematous laryngitis and tracheitis in tracheotomized patients may be almost as troublesome. Patients with subglottic edematous obstructive laryngitis can enjoy relief and progress favorably for several days after tracheotomy; then the airways will become obstructed with thick exudate and the treatment described in the preceding paragraph will be required. Inasmuch as the transition is incident to secondary infection, cases in which this occurred are not included in our table under the heading of "acute obstructive laryngotracheobronchitis."

The therapeutic value of the steam tent cannot be overestimated, and in the event of coexisting pneumonia, oxygen may be administered simultaneously with steam.

SUMMARY AND CONCLUSIONS

There is apparent uncertainty concerning croup, particularly from the point of view of definition, classification, diagnosis and treatment.

We consider it necessary to define the condition properly and to classify the cases correctly. It is important to check clinical observations with minute anatomic and pathologic studies.

Laryngoscopic examination was performed in all the cases of our series in which there was doubt as to the pathologic evidence, prognosis or therapy. We found it necessary to use the laryngoscope in more than 30 per cent of our cases.

The presence or absence of supraglottic edema and the position and motion of the vocal cords before and after suctioning of the subglottic area and trachea have furnished us a dependable guide to the choice of therapy.

For croup of the supraglottic obstructive type, tracheotomy is the logical treatment. For that of the subglottic exudative type, suction is adequate. For that of the subglottic edematous type, tracheotomy is preferable. In acute obstructive laryngotracheobronchitis, tracheotomy followed by repeated bronchoscopic suctioning through the wound is necessary. Intelligent postoperative care of patients with croup of this type is most important.

It is generally accepted that close cooperation among the pediatrician, the laryngologist and members of the house and nursing staffs helps to increase the efficiency of the care of patients with this disease and tends to moderate the death rate.

The incidence of morbidity and mortality is affected by the occurrence of Staphylococcus aureus haemolyticus as well as by complicating bronchopneumonia, which was present in 25 per cent of our cases.

Subglottic edematous obstructive laryngitis and acute obstructive laryngotracheobronchitis are most frequently complicated by pneumonia.

A state of exhaustion from prolonged air hunger must be carefully differentiated from so-called toxic diphtheria. Measures directed to the relief of laryngeal obstruction are not contraindicated by coexisting pneumonia.

A new charting system for the recording of data on patients with croup is recommended.

The cases included in this survey are from the pediatric services of Drs. Harold L. Barnes, Thurman B. Givan, Murray B. Gordon, A. M. Litvak, Benjamin Kramer, Kenneth Jennings and B. Newman.

The investigation was conducted by the rhinolaryngologic staff of Dr. Harry Meyersburg. Drs. Juluis A. E. Sass and Julius Belinkoff, of the pathologic department, cooperated in the study. Dr. A. V. Shapiro, roentgenologist, and Dr. Henry Feinberg, superintendent, as well as members of the medical board and of the house staff, especially Dr. Samuel Stone, and members of the nursing staff of the Kingston Avenue Hospital assisted in the work.

SIGNIFICANCE OF THE p_H OF NASAL SECRETIONS IN SITU

FURTHER STUDIES

NOAH D. FABRICANT, M.D.
CHICAGO

In a previous communication I [1] maintained that nasal vasoconstrictors with an unusually high p_H value may (1) disturb the normal p_H level of nasal secretions in situ in the clinically normal nose, (2) enhance irritation of the nasal mucous membrane and (3) postpone the return of the nasal mucosa from a pathologic alkaline status to a normal, slightly acid status. It has been suggested that during acute rhinitis, acute rhinosinusitis and the more active stages of allergic rhinitis the employment of a nasal vasoconstrictor which lowers the alkaline p_H to a normal acid level, between 5.5 and 6.5, is most desirable. The fact that 1 per cent ephedrine hydrochloride in physiologic solution of sodium chloride—a nasal vasoconstrictor almost universally acceptable to rhinologists—has a p_H (5.9) that practically cleaves the midpoint between 5.5 and 6.5 furnishes adequate testimony to the truth of this concept.

It has been suggested further that there should be a standardization of the p_H values of the various nasal vasoconstrictors sold to the public. The generally accepted American advertising slogan, "It is time to alkalinize," certainly does not apply to the treatment of the nasal mucous membrane. The return of the nasal mucous membrane to a normal, slightly acid status is accomplished—unwittingly or not—by every physician who administers—and I mention but a few of the agents—1 per cent ephedrine hydrochloride in physiologic solution of sodium chloride and such ephedrine-containing preparations as glucofedrin and isohalant. Each of these vasoconstrictors has a p_H in the acid range, and each is designed to restore and maintain normal ciliary activity. A normal, slightly acid status, however, cannot be induced by nasal vaso-

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This is part 2 of the work that has received the Casselberry Award of the American Laryngological Association for 1941.

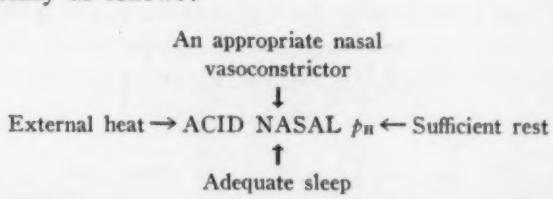
1. Fabricant, N. D.: Significance of the p_H of Nasal Secretions in Situ, Arch. Otolaryng. **33**:150 (July) 1941.

constrictors which, because of their inherently high p_H, superimpose additional alkalosis on an already abnormal alkaline nasal mucous membrane.

Recent experiments performed by Dr. William F. Petersen, professor of pathology at the University of Illinois College of Medicine, in the field of my own investigations indicate to me that there is a definite relationship between well established therapeutic measures employed against acute rhinitis and the concept I have proposed, that of the institution of a physiologic restoration to a normal, slightly acid nasal p_H. Constant and continuous observation of a clinically normal subject for twenty-four hours will show that the p_H of nasal secretions in situ varies with rest and sleep, diurnally and nocturnally.

Prolonged rest and sleep promote an acid trend in the nasal p_H. The nasal p_H range is more acid during the night than it is during the day. That the trend toward acidity under these conditions is not an isolated phenomenon peculiar to the nose has been demonstrated by Grossman and Brickman.[2] These investigators, examining hourly over a twenty-four hour period the saliva of 36 persons, found a p_H range indicating that the saliva is more acid at night than it is during the day. Finally, I have observed that a distinct drift toward acidity in nasal secretions is produced within twenty minutes by the administration of external heat to the face.

The implications of this correlated group of observed phenomena become obvious at once, particularly when one considers that acceptable nasal therapy for acute rhinitis and acute rhinosinusitis includes (1) the careful selection and administration of an appropriate nasal vasoconstrictor, (2) the assurance of sufficient rest and adequate sleep and (3) in selected cases of acute nasal sinusitis, the judicious administration of external heat for the relief of pain. This therapy may be expressed diagrammatically as follows:



2. Grossman, L. I., and Brickman, B. M.: Comparison of Nocturnal and Diurnal p_H Values of Saliva, J. Dent. Research **16**:179-182 (June) 1937.

Each of these meritorious therapeutic measures produces uniformly a single nasal p_H phenomenon—acidity! However, should an occasional therapeutic nihilist, rising to exclaim that the use of nasal vasoconstrictors may do more harm than good, omit this specific measure—the remaining measures, which he certainly must countenance, will still produce a nasal p_H in the acid range.

EFFECT OF TOPICAL ANESTHETICS

By utilizing a silver–silver chloride glass electrode in conjunction with the Coleman electrometer and by conducting minute to minute readings of the p_H values of nasal secretions in situ for periods of one to one and one-half hours, it is possible to interpret satisfactorily the role of topical anesthetics in elective nasal operations. The most commonly employed topical anesthetic agents manufactured in the United States have been studied in a lengthy series of experiments. The following agents were surveyed:

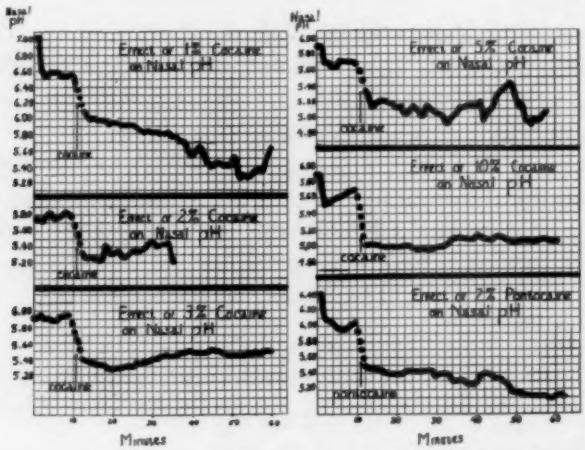
	p_H
2 per cent butyn sulfate	6.0
2 per cent pontocaine hydrochloride	5.4
1 per cent to 10 per cent cocaine	3.32 to 5.19
1 per cent diothane hydrochloride	4.5
1 per cent to 10 per cent metycaine	4.38 to 4.4

Some rhinologists prefer to add to a topical anesthetic solution a few drops of an epinephrine solution for the vasoconstrictor effect. Two widely used epinephrine solutions are:

	p_H
Suprarenin (synthetic epinephrine bitartrate) 1:1,000	3.7
Adrenalin chloride (epinephrine hydrochloride made from the adrenal gland) 1:1,000	2.3 to 2.5

It will be observed that the p_H of each topical anesthetic agent and of each epinephrine solution falls specifically within the acid range. Since the manner in which the trend of the nasal p_H is affected depends entirely on the p_H of the drug or the combination of drugs employed, it is apparent that each topical anesthetic—with or without the addition of several drops of an epinephrine solution—produces a noticeable drift in the nasal reaction toward increased acidity. This is well illustrated by the effect on nasal p_H produced by cocaine solution, in strengths of 1, 2, 3, 5 and 10 per cent and by pontocaine hydrochloride 2 per cent as shown in the chart.

Since the competent rhinologist who is to perform a nasal operation elects a time when the patient is free from any acute inflammatory nasal condition—that is, when the patient's nasal p_H lies within the normal, slightly acid range between 5.5 and 6.5—it is clear that the operation if done with the use of any of the topical anesthetics enumerated must be performed in a completely acid nasal environment. For under circumstances such as these (1) the p_H value of the nasal secretions in situ is below 7, (2) the topical anesthetic agents are acid, (3) the vasoconstrictors are acid and (4) the noticeable effect of the topical anesthetics and vasoconstrictors, alone or in combination, is acid.



The drift toward increased acidity in nasal environment produced by cocaine solution in strengths of 1, 2, 3, 5 and 10 per cent and 2 per cent pontocaine hydrochloride.

COMMENT

What is the significance of these observed phenomena? It will be recalled that my earlier investigations concerned themselves with the selection of an appropriate nasal vasoconstrictor, one which (1) is designed to restore and maintain normal ciliary activity but which (2) also is slightly acid, having a p_H value preferably between 5.5 and 6.5. I believe that an excellent example of this type of nasal vasoconstrictor is the highly praised 1 per cent ephedrine hydrochloride in physiologic solution of sodium chloride, with a p_H of 5.9.

The generally recognized therapeutic measures for patients with acute rhinitis or acute rhinosinusitis include sufficient rest, adequate sleep and the judicious administration of external heat for the relief of pain. I have shown that each of these acceptable measures produces uniformly a single nasal p_H phenomenon—acidity. However, should a patient require a nasal

operation to be performed later—when the acute symptoms have disappeared and the nasal p_H range has been restored to a normal, slightly acid status—this operation at the elective time will be performed in a completely acid nasal environment.

In view of the accumulated and mounting evidence concerning the significance of the p_H of nasal secretions in situ, it is suggested that every nasal vasoconstrictor prescribed by physicians and sold to the public should satisfy two basic physiologic requirements: (1) It should restore and maintain normal ciliary activity and (2) it should be slightly acid, with a p_H value that is in the normal p_H range of nasal secretions in situ, i. e., between 5.5 and 6.5. To emphasize the ciliary factor apart from the p_H factor is to tell but half of the story of applied nasal physiology.

SUMMARY

Acceptable nasal therapy for acute rhinitis and acute rhinosinusitis includes (1) the careful selection and administration of an appropriate nasal vasoconstrictor, (2) the assurance of sufficient rest and adequate sleep and (3) the judicious administration of external heat for the relief of pain in selected cases of acute nasal sinusitis. Each of these well recognized therapeutic measures produces uniformly a single nasal p_H phenomenon—acidity.

All elective nasal surgical procedures which require the use of topical anesthetics and vasoconstrictors are performed in a completely acid nasal p_H environment: (1) The p_H of the nasal secretions in situ is below 7; (2) the topical anesthetic agents are acid; (3) the vasoconstrictors are acid and (4) the noticeable effect of the topical anesthetics, alone or in combination with a few drops of a vasoconstrictor, is acid.

The return of the nasal p_H to an acid status is accomplished—unwittingly or not—by every physician who administers 1 per cent ephedrine hydrochloride in physiologic solution of sodium chloride and preparations of ephedrine, such as gluco-fedrin and isohalant. Should an occasional therapeutic nihilist omit the use of a nasal vasoconstrictor but utilize such measures as adequate rest and sleep and external heat—singly or collectively—the end result is only that of establishing an acid nasal environment.

It is suggested that every nasal vasoconstrictor prescribed by physicians and sold to the public should satisfy two basic physiologic requirements: (1) It should restore and maintain normal ciliary activity and (2) it should be slightly acid with a p_H value in the normal p_H range of nasal secretions in situ, i. e., between 5.5 and 6.5. To emphasize the ciliary factor apart from the p_H factor is to tell but half the story of applied nasal physiology.

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EFFECT OF SILVER PREPARATIONS AND ANTISEPTICS ON THE p_H OF NASAL SECRETIONS IN SITU

NOAH D. FABRICANT, M.D.
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I have suggested[1] that every nasal vasoconstrictor prescribed by physicians and sold to the public should satisfy two basic physiologic requirements: (1) It should restore and maintain normal ciliary activity and (2) it should be slightly acid, with a p_H value in the normal p_H range of nasal secretions in situ, i. e., between 5.5 and 6.5. To emphasize the ciliary factor apart from the p_H factor is to tell but half the story of applied nasal physiology.

I have demonstrated the significance of the p_H factor by showing that each of the well recommended therapeutic measures employed in the treatment of acute rhinitis and acute rhinosinusitis—namely, the careful selection and administration of an appropriate nasal vasoconstrictor, the assurance of sufficient rest and adequate sleep and the judicious administration of external heat—produces uniformly a single nasal p_H phenomenon, acidity. Further, I have maintained that any nasal surgical procedure which is performed at an elective time and with the use of topical anesthetics, alone or in combination with a few drops of a vasoconstrictor, will be performed in a completely acid nasal environment.

In evaluating the effect of various commonly employed silver preparations and antiseptics on the nasal secretions in situ, I concern myself only with the p_H factor. All the agents I have studied, with the exception of one silver preparation and one antiseptic, are alkaline. Hence when I quote the investigations of other workers with regard to the ciliary factor, it should be borne in mind that for the most part their investigations were conducted with alkaline silver preparations and alkaline antiseptics. At present I shall not attempt to state what effect silver preparations and antiseptic solutions—theoretically possessing a p_H between 5.5 and 6.5—have on the ciliary factor. It is quite con-

From the Department of Laryngology, Rhinology and Otology and the Department of Pathology, University of Illinois College of Medicine.

This is part 3 of the work that has received the Casselberry Award of the American Laryngological Association for 1941.

1. Fabricant, N. D.: Significance of the p_H of Nasal Secretions in Situ: Further Studies, Arch. Otolaryng., this issue, p. 297.

ceivable, however, that the adjustment of these preparations to a p_H value approximating the normal p_H range of nasal secretions in situ will bring about an entirely new conception of their relationship to the ciliary factor.

Lierle and Moore [2] asserted that mild protein silver in concentrations of 5, 10 and 20 per cent produces an initial speeding of ciliary activity, which is followed by a slowing. They stated the belief that this may be due to the water solvent rather than to the drug. Proetz [2b] observed no effect on the cilia from the application of 10 per cent mild protein silver but noted an interference with the stream of mucus, which he interpreted as due to clumping produced by the precipitated drug. Finally, in their thoughtful discussion of the problem of intranasal medication, Walsh and Cannon [3] wrote that although some of the so-called antiseptics, e. g., mild protein silver, cause no apparent harm to ciliary activity, their antiseptic value is doubtful.

In my investigation of the effect of silver preparations and antiseptics on the p_H of nasal secretions in situ I have continued to use a silver–silver chloride glass electrode in conjunction with the Coleman electrometer. The method has been described in detail elsewhere.[4] Minute to minute readings of the p_H values of nasal secretions in situ were taken for periods of one to one and one-half hours. Some of the more common silver preparations employed by American rhinologists in nasal therapy were studied in a detailed series of experiments. The following were the silver preparations surveyed:

	Strength	p_H
Mild protein silver U.S.P.		
(a) Silvol	5% to 20%	8.0
(b) Solargentum	5% to 20%	9.5 to 9.7
(c) Argyrol	5% to 20%	9.1 to 9.2
(d) Silver nucleinate	5% to 20%	9.5 to 10.0
Strong protein silver U.S.P.		
(a) Protargol	0.25% to 1%	8.2 to 8.8
Colloidal silver iodode		
(a) Neo-silvol	5% to 20%	7.5
Colloidal silver bromide		
(a) Argental	5% to 20%	4.7 to 4.8

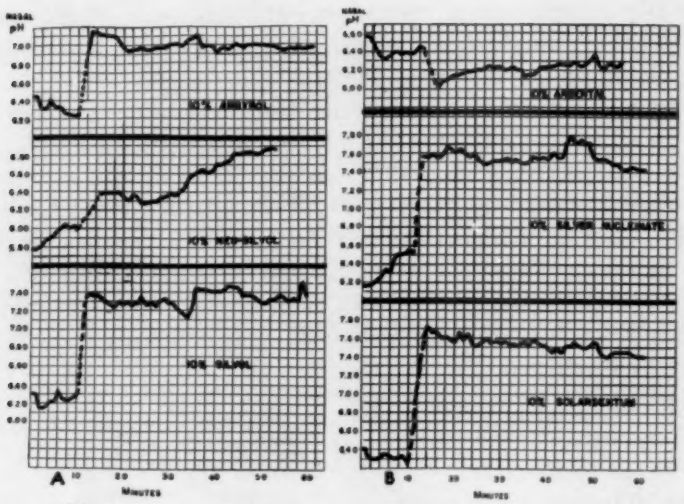
It will be observed that the p_H of six of the seven silver preparations studied falls specifically within the alkaline range. The sole exception—

2. (*a*) Lierle, D. M., and Moore, P. M.: Effects of Drugs on Ciliary Activity of Mucosa of Upper Respiratory Tract, Arch. Otolaryng. **19**:55 (Jan.) 1934. (*b*) Proetz, A. W.: Ann. Otol., Rhin. & Laryng. **43**:450, 1934.

3. Walsh, T. E., and Cannon, P. R.: Ann. Otol., Rhin. & Laryng. **47**:579, 1938.

4. Fabricant, N. D.: Significance of the p_H of Nasal Secretions in Situ, Arch. Otolaryng. **33**:150 (July) 1941.

argental—has a p_H below the normal p_H range of nasal secretions in situ and differs from other colloidal silver compounds in that it consists essentially of silver bromide and not of silver oxide. Since the manner in which the trend of the nasal p_H is affected depends entirely on the p_H of the drug employed, it is apparent that the six alkaline silver preparations produce a noticeable drift upward in the nasal p_H, i. e., toward alkalinity. On the other hand, the sole acid silver preparation produces a characteristic drift downward in the nasal p_H, i. e., toward increased acidity. None of the silver preparations permits the normal fluctuation of nasal p_H to pursue the relatively even tenor of its way. Excellent illustrations are the effects on nasal p_H that are produced by 10 per cent argyrol,



A, the drift toward alkalinity in nasal secretions in situ produced by 10 per cent argyrol, 10 per cent neo-silvol and 10 per cent silvol. *B*, the drift toward increased acidity in nasal secretions in situ produced by 10 per cent argental and the characteristic trend toward alkalinity produced by 10 per cent silver nucleinate and by 10 per cent solargentum.

10 per cent neo-silvol, 10 per cent silvol (fig. 1 *A*) and by 10 per cent argental, 10 per cent silver nucleinate and 10 per cent solargentum (fig. 1 *B*).

The aforementioned observations hold clinical significance, for it is believed that those silver preparations which either enhance or perpetuate the alkalinity of the nasal secretions in acute rhinitis and acute rhinosinusitis prolong an undesirable status—an alkaline status in which the bacteria producing the acute inflammation find a fertile field for growth. The numbers of bacteria that I have been able to recover from the nasal mucous membrane at high and low points in the normal fluctu-

ation of the nasal p_H often differ astonishingly. For the most part, fewer bacteria are recovered at the low (more acid) phase than at the high (less acid) phase. This supports the view that the bacteria producing acute rhinitis grow best in an alkaline milieu. It is more than likely that when there is alkalosis of the nasal mucous membrane the changes in capillary, arterial and membrane permeability proceed at a tempo sufficient to prolong an abnormal physiologic nasal environment.

It is therefore suggested that during acute rhinitis and acute rhinosinusitis the employment of a silver preparation which lowers the high p_H to a level between 5.5 and 6.5—approximating the normal p_H status of nasal secretions in situ—is most desirable. An ideal silver preparation, no less than a proper nasal vasoconstrictor, should not only restore and maintain normal ciliary activity but also be slightly acid with a p_H value between 5.5 and 6.5. It is quite conceivable that such a p_H improvement in the silver preparations would bring about an entirely new conception of the relationship of these preparations to the ciliary factor.

EFFECT OF ANTISEPTICS

In evaluating the effect of antiseptic solutions on the nasal secretions in situ, I again must concern myself only with the p_H factor. Of the four solutions I have studied, three—metaphen, merthiolate and mercurochrome—are quite alkaline. The one exception, mercresin, is slightly acid, its p_H falling within the normal p_H range of nasal secretions in situ. The observations of other investigators [2] with regard to the ciliary factor reveal that merthiolate 1 : 1,000 to 1 : 5,000 causes an initial slowing of ciliary action, with cessation after a few minutes, and that 2 per cent mercurochrome is definitely detrimental to ciliary activity.

Four antiseptic solutions were studied for an explanation of their effect on the p_H of nasal secretions in situ. The strengths of these solutions used in my experiments are those recommended by the pharmaceutic manufacturers. The antiseptic solutions studied were:

	p_H
Metaphen 1:2,500 to 1:500	10.0 — 11.0
Merthiolate 1:1,000 to 1:5,000	9.6 — 10.0
Mercurochrome 0.25 per cent to 2 per cent	8.8 — 9.8
Mercresin diluted 1:5	5.5 — 5.7

In accordance with what appears to me to be a universal law—i. e., that alkaline solutions instilled into the nose attract the nasal p_H toward a characteristic alkaline status while acid solutions draw the nasal p_H toward a definitely acid status—I find that metaphen, 1 : 2,500 and 1 : 500, merthiolate 1 : 1,000 and 1 : 5,000 and mercurochrome 0.25 to 2 per cent, convert the normal, slightly acid nasal p_H status to an alkaline status. The use of these drugs during acute rhinitis and acute rhinosinusitis

would superimpose additional alkalosis on an already abnormal alkaline nasal mucous membrane. This I believe to be undesirable. It is quite possible, however, that one of the reasons—surely there are others in addition to that relating to the ciliary factor—why these three antiseptic solutions have been found to be ineffectual in the therapy of acute rhinitis is this: They are inherently alkaline. Mercresin in a dilution of 1:5 satisfies the p_H standards I have advanced as appropriate for nasal medicaments. It has a p_H of 5.5 to 5.7, a value which falls within the normal p_H range of nasal secretions in situ. Just what relationship this antiseptic has to the ciliary factor can become the subject for further experimentation.

SUMMARY

Silver preparations which either enhance or perpetuate the alkalinity of the nasal secretions found in acute rhinitis and acute rhinosinusitis prolong an undesirable nasal status—an alkaline status, in which the bacteria producing the acute inflammation find a fertile field for growth. It is suggested that during acute rhinitis and acute rhinosinusitis the employment of a silver preparation which lowers the p_H to a level between 5.5 and 6.5 is most desirable. Silver preparations standardized to a p_H level between 5.5 and 6.5 could produce this effect. It is quite conceivable that such a p_H improvement in the silver preparations would bring about an entirely new conception of their relationship to the ciliary factor.

Because of their inherently alkaline composition, metaphen 1:2,500 and 1:500, merthiolate 1:1,000 and 1:5,000 and mercurochrome 0.25 to 2 per cent, convert the normal slightly acid nasal status to an alkaline status. During acute rhinitis and acute rhinosinusitis, such antiseptic solutions superimpose additional alkalosis on an already abnormal, alkaline nasal mucous membrane. This is believed to be undesirable. Mercresin in a dilution of 1:5 satisfies the p_H standards I have advanced by having a p_H value that falls within the normal p_H range of nasal secretions in situ. The exact relationship of this antiseptic to the ciliary factor can become the subject for further investigation.

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THE DEFORMED NOSE

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HISTORICAL NOTES

Rhinoplastic surgery is not a new art. It was practiced thousands of years ago by the Hindus in India, where criminals had their noses cut off as a punitive measure. Even in those ancient times the art of the plastic surgeon was indispensable. At the beginning of the Christian era, surgeons were shifting tissues from the forehead for plastic repair of the nose, around which organ most plastic surgical procedure has centered. In the second century lost portions of the nose were being crudely restored, and by the sixteenth century the ingenious Italian Tagliacozzi was performing rhinoplastic operations with an amazing skill for the period. Surgeons of America and Europe who followed him became enthusiastic over the plastic art and developed it to its present state.

The World War was responsible for the greatest impetus plastic reparative surgery has ever received. The brilliant results obtained by plastic surgeons during and since that conflict have also served as an impetus to many surgeons to turn to this field and have caused the public to be conscious of the great technical improvements which have been made in plastic surgical procedures, in the past score of years and of the esthetic usefulness of these procedures.

SOCIAL ASPECTS OF PLASTIC SURGERY

Since automobiles have crowded the roads, thus increasing the number of accidents, and with the advent of high-powered machinery and the airplane and other agents of "streamlined speed," there has been an increase in the incidence of facial injuries. The nose is an organ of esthetic importance, and its prominence on the face results in its being most ordinarily offended in facial injuries. Disease, congenital defect or injury involving the nose obviously is most detrimental to one's appearance and health. Frequently persons withdraw from normal social intercourse because of such "stigmas." These unfortunate people

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rapidly become subject to inferiority complexes which can easily be avoided, for such nasal deformities, which are social and professional handicaps and result in discrimination against the handicapped person, are amenable to correction. Of all the plastic operations about the face, none have made so great strides as have the rhinoplastic procedures.

RHINOPLASTIC SURGERY DEFINED

Corrective rhinoplastic surgery is that specialty which deals with the correction of nasal defects, congenital or acquired, with the aim of improving both function and esthetic effect. Keeping in mind the importance of function, since health is of primary importance and beauty secondary, the rhinoplastic surgeon must strive toward both in each operative procedure. Therefore, it is obvious that this specialty is an art as well as a science and that the scope and problems are of great magnitude. The object of this paper is to describe typical cases encountered in the practice of rhinoplastic surgery and the nasal deformities amenable to treatment and to demonstrate what may be expected in the correction of disfigurements about the nose by partial or complete endonasal rhinoplasty. I have purposely omitted from this report all cases in which skin grafts were required for loss of nasal substance, as that subject would necessitate a separate lengthy dissertation. I should like to mention, however, that when it becomes necessary to use skin flaps or free grafts for nasal coverings the optimal donor areas are the upper eyelids and the postauricular regions, together with the inner aspect of the arm. The forehead also offers an ideal skin for matching. The most meticulous technic and patience for detail are paramount in matching and grafting in these regions. The illustrations elucidate the technic employed.

PROGRAM OF RECONSTRUCTION

There are three distinct periods or phases of nasal reconstruction: the preoperative period, the operation itself and the postoperative treatment. In the first phase, great care is required to rule out the presence of systemic disease, coryza or sinusitis. If any of these conditions are present, they must be rectified before correction is attempted. Also during this phase, photographs are taken, showing the nose in profile, from the front, and with the patient's head tilted back to show the nasal introitus and tip. The photographs can be augmented by the use of plaster casts of the face. These furnish an excellent means of preoperative determination of the approach and procedure necessary and incidentally are most helpful in the event of legal complications.

Local anesthesia (2 per cent procaine hydrochloride with 30 drops of a 1:1,000 solution of epinephrine hydrochloride to the ounce) is used almost exclusively. Preoperative sedation is accomplished with a sub-

stantial dose of a barbiturate (6 grains [0.39 Gm.] sodium amytal or 3 grains [0.19 Gm.] pentobarbital sodium) given one and one-half hours before the operation. If the patient is not sufficiently composed thirty minutes before the operation, a narcotic is given (0.17 grain [0.01 Gm.] morphine sulfate with 0.0067 grain [0.0004 Gm.] scopolamine hydrobromide). The use of narcotics is to be avoided, as it frequently causes vomiting and thus the passage of the contents of the stomach through the nose. Sterilization of the skin is obtained by the application of liniment of soft soap U. S. P. and metaphen.

In all nasal corrections, only the endonasal route is used and all incisions are intranasal. The operative field should be well illuminated without glare to prevent ophthalmic fatigue when lengthy detailed plastic procedures are attempted. The operating personnel should be well trained as a team, and absolute cooperation between the surgeon and his assistants and nurses is indispensable. Unnecessary conversation and noises are to be avoided, as the patient is conscious and under nervous strain.

In all cases, immediately after the operation the nose is splinted externally with Stent's composition molded over soft flannel and retained with adhesive strips. The cast remains on the nose for five days. The mild pressure prevents edema and the formation of hematomas and immobilizes the nasal bones. Postoperative edema of the eyelids is treated by the application of cotton pledgets saturated with iced boric acid solution. Any sutures used are removed on the fifth day. All packings are removed after thirty-six hours. After the cast has been removed, the Joseph [1] nasal brace is applied for one hour daily for six weeks when necessary, to assure the proper width of the nasal bridge.

TYPICAL NASAL DEFORMITIES

Among the common deformities are (1) deflected nose, (2) hump nose, (3) elongated nose, (4) bulbous nasal tip, (5) saddle nose and (6) combined deformity of the nose and chin. These may occur separately or in combinations. For example, the nose may be elongated and twisted, with a bulbous tip, or a hump nose may have no other deformity. The correction of each deformity is the same, whether it occurs singly or in combination.

Deflected Nose (fig. 1).—A deflection of the nose may be congenital, but it is most often caused by trauma, usually an old fracture.[2] More

1. Joseph, J.: Nasenplastik und sonstige Gesichtsplastik, Leipzig, Curt Kabitzsch, 1928.

2. Straith, C. L., and DeKleine, H.: Modern Management of the Fractured Nose: Collective Review, Internat. Abstr. Surg. **66**:9, 1938; Surg., Gynec. & Obst., January 1938.

often than not there is a concurrent nasal obstruction from septal displacement. If the deflection is of a minor degree, refracture of the nasal bones from the nasal processes of the superior maxillas will suffice. This is most accurately done with an angular Joseph [1] saw (fig. 1, *2*). This refracture is greatly facilitated if the osteotome (fig. 2, *13*) is

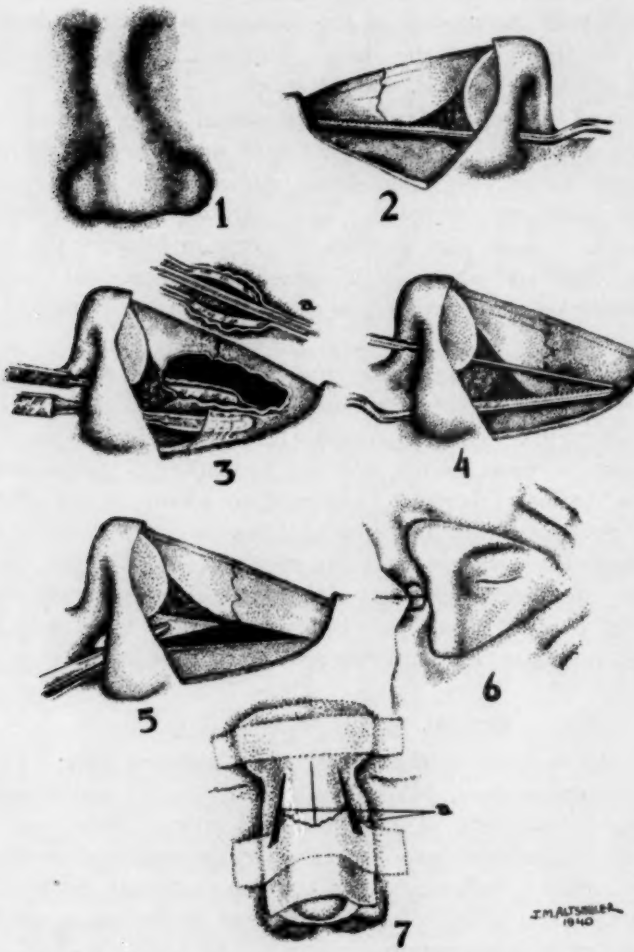

Fig. 1.—Steps in the reconstruction of a deflected nose: (*1*) the deformity; (*2*) simple bone incision on the convex side; (*3*) elevation of the periosteum externally and the mucous membrane internally (*a* indicates the periosteum); (*4* and *5*) excision and removal of a triangle of bone on the concave side; (*6*) nasal fracture into the midline, and (*7*) the method of postoperative fixation (*a* indicates site of bone incisions).

used after the bony arch has been sawed through. This completely immobilizes the nasal bones (fig. 2, *15*). If the degree of nasal deflec-

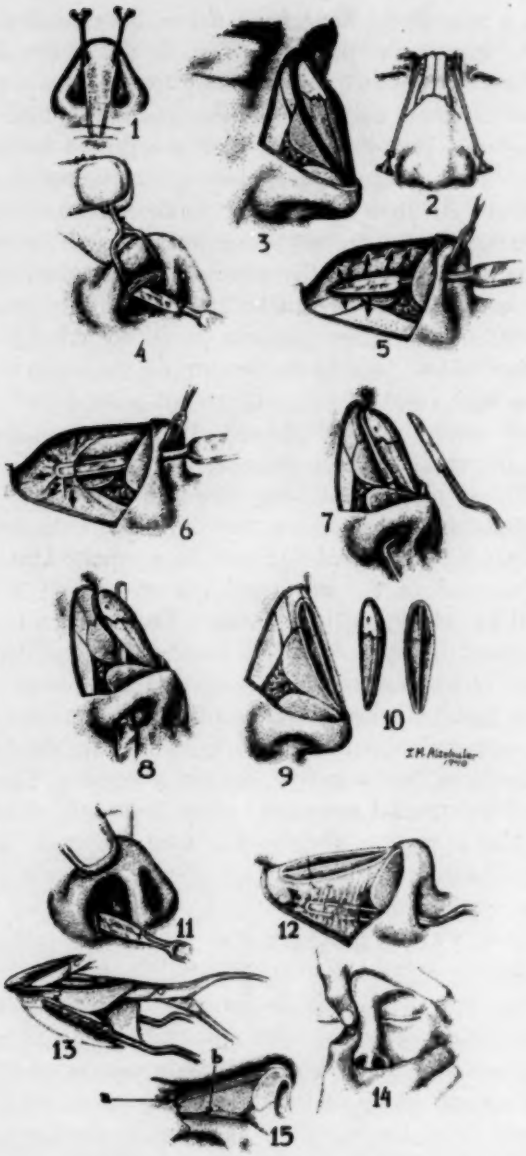

Fig. 2.—Plastic reconstruction of a typical hump nose: (*1* and *2*) four points for the introduction of anesthetics; (*3*) the deformity and the area to be excised; (*4*) the incision; (*5*) undermining of the skin; (*6*) undermining of the periosteum; (*7* and *8*) removal of the hump; (*9*) the hump excised, with wide bridge visible; (*10*) osteocartilaginous "hump"; (*11*) skin incision for narrowing of the nose; (*12*) the periosteum elevated; (*13*) the saw in position for bone incision in the frontal process of the maxilla, while the bones are immobilized at the nasofrontal suture by means of an osteotome; (*14*) fracture of the frontal process, and (*15*) the bridge narrowed (*a* indicates the fracture at the nasofrontal suture and *b* the bone incision in the frontal process of the maxilla).

tion is great, a triangle of bone from the wider side of the nose must be removed to correct the deformity (fig. 1, *5*). Five days after the operation, a brace is applied daily, making pressure on the convex side of the nose only so as to assure reposition into the midline. Deflections of the cartilaginous portions of the nose are caused by torsion of the nasal septum. If they are severe, they are corrected by partial submucous resection. In the correction of minor deflections, the septum is completely immobilized from its attachments to the lateral cartilages, the nasal spine, the vomer and the columella and reposed into its normal position.[3] It is maintained in situ by appropriate intranasal and extranasal supports.[4] For better support it is sometimes necessary to resuture the lateral cartilage to the septum on the concave side. Intranasal packing with oiled finger cots packed with 5 per cent bismuth tribromphenate gauze is used. Externally, a splint of Stent's composition retains the nasal bones in their normal position (fig. 1, *7*). The septal resection is of greatest importance in rhinoplastic surgery. I cannot overemphasize the necessity that only the bent, obstructing portion of the septum be removed and that an adequate buttress be left to support the dorsum of the nose and the nasal tip. Otherwise one deformity will be substituted for another. Overzealous resection of the septum will result in either a saddle nose with a flat dependent nasal tip or a "false" hump created by the normal nasal bone, owing to the collapse of the septal portion of the profile. If the precaution of leaving an adequate support is not taken, simple removal of the hump becomes extremely hazardous, lest a saddle deformity result. The nasal plastic procedure and submucous resection, when indicated, should always be combined at one operation, for the following reasons: 1. The double expense and discomfort of two operations are obviated. 2. When all tissues are exposed at one operation, it is infinitely easier to accomplish the dual purpose without excessive removal of bone and cartilage, and the two procedures become complementary.[5]

Hump Nose (fig. *2*).—This deformity, sometimes called hook nose, is due to overdevelopment and superabundance of the supporting structures of the nose. This is the most commonly seen nasal deformity. The formation of a nasal hump may be caused by excessive prominence of the nasal bones or of the septal cartilage or an amalgamation of both,

3. Metzenbaum, M.: (*a*) Dislocation of the Lower End of the Nasal Septal Cartilage, Arch. Otolaryng. **24**:78 (July) 1936; (*b*) Replacement of Lower End of Dislocated Septal Cartilage Versus Submucous Resection of Dislocated End of the Septal Cartilage, ibid. **9**:282 (March) 1929.

4. Watkins, A. B. K.: Notes on Nasal Plastic Surgery, J. Laryng. & Otol. **48**:809 (Dec.) 1933.

5. Salinger, S.: Deviations of the Septum in Relation to the Twisted Nose, Arch. Otolaryng. **29**:520 (March) 1939.

which is the most usual. After being freed by means of the blunt end knife from its attachment to the cartilaginous septum, the nasal hump is removed subperiosteally with bayonet saws at exactly the same level on both sides (fig. 2, *7*). It is withdrawn with grasping forceps. After the hump is removed, the bony arch is narrowed (fig. 2, *15*). These nasal humps furnish useful transplant material for the correction of depressions of a similar size over bony areas, such as that seen over the site of collapse of a frontal sinus after a radical operation, and also for the correction of moderately receding chins.

Elongated Nose (fig. 3).—The correction of an elongation of the nose is accomplished by simple excision of a wedge-shaped portion of the free end of the nasal septum, full thickness, with the apex posterior. The size of the section removed depends on the amount of shortening desired. Occasionally the procedure is hampered by the presence of a short columella. When the requisite amount of shortening cannot be procured otherwise, it becomes necessary to lengthen the columella. After the septum is resutured to the columella, the nasal tip is raised (fig. 3, *7*). If the lateral cartilages overlap, they are trimmed (fig. 3, *6*). This procedure also corrects an accompanying dislocation of the end of the septum in an elongated nose (fig. 3, *5*). If shortening is not necessary, such a dislocation is corrected by a different technic.[3a] It is now known conclusively that an elongation of the nose with a distortion of the tip disrupts the normal route of inspired air,[6] producing eddies and whorls in the nasal passages, which cause dryness of the nose, headaches, frequent colds and stuffiness. The shortening process described here can eradicate these conditions.

Bulbous Nasal Tip (fig. 4).—The most common deformity of the tip, a bulbous contour, may accompany any of the other nasal deformities. The procedure depends on which portions of the alar cartilages are deformed. The approach used by me is original and has been used satisfactorily in many cases for the past five years. It differs from the Joseph [1] technic, which is most widely employed. It excludes the use of the punch for removal of the alar cartilage and also excludes the incision along the margin at the lower end of the alar cartilage. This marginal incision, which is widely used, frequently produces unnecessary contractures, adhesions and irregularities of the edge of the nostril.

Technic (fig. 2).—An intercartilaginous incision is made between the lateral and the alar cartilage; a second incision is made through the alar cartilage, parallel to the nasal septum, and carried forward to the edge of the nostril. The alar cartilage is then undermined thoroughly, and the skin is allowed to redrape itself easily over the reshaped cartilaginous framework. The cartilage is everted from

6. Proetz, A. W.: Some Intimate Studies of Nasal Function: Their Bearing on Diagnosis and Treatment, Ann. Otol., Rhin. & Laryng. **41**:125 (March) 1932.

above downward and presented for trimming. Sections of it are then removed, with the nasal lining, at the precise sites of the maximum bulbosity of the tip, whether it be the medial crus, the crus apicis or the lateral crus. By this method the edge of the nostril is unimpaired, because the marginal incision is not used, and thus contractures and adhesions in juxtaposition to the columella are elimi-

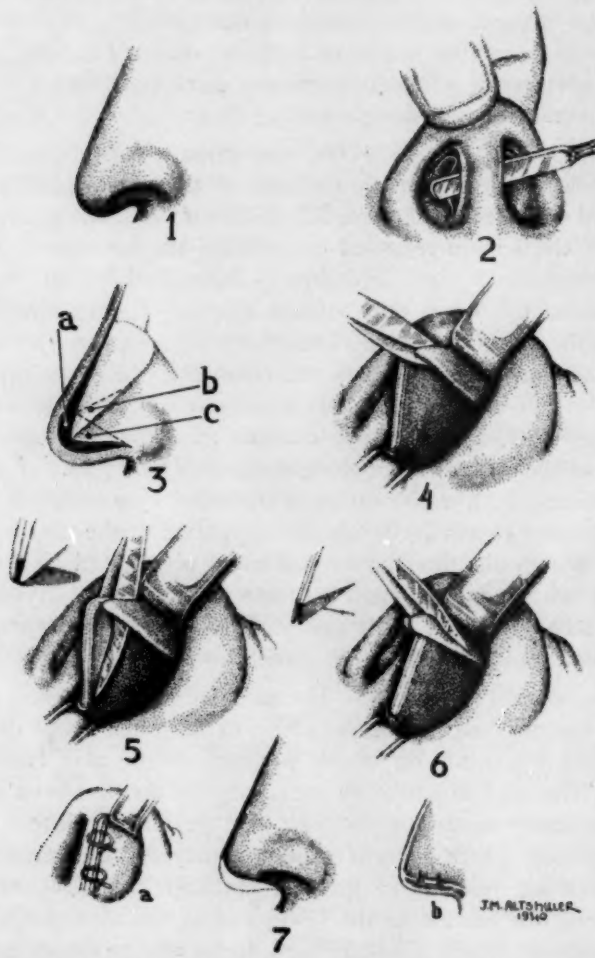

Fig. 3.—Method of shortening an elongated nose: (*1*) the deformity; (*2*) separation of the septum from the columella; (*3a*) the skin undermined and (*b* and *c*) areas of septum and lateral cartilage removed; (*4, 5* and *6*) method of removal of the septum and lateral cartilage, and (*7*) the results after the septum has been sutured to the columella (*a* indicates the space created when the skin is separated by undermining from the nasal framework and *b* the section of lateral cartilage to be removed).

nated. A light packing of bismuth tribromphenate gauze is introduced at the site of the excised alar cartilage and maintained there for thirty-six hours by strips

of adhesive tape, which is lined on its under surface, around the tip. This produces the much desired effect of pointing a tip that was previously bulbous (fig. 4, *6*).

Saddle Nose (fig. 5).—Simple, uncomplicated saddle nose is most frequently caused by trauma,[7] which results in a depression of the sup-

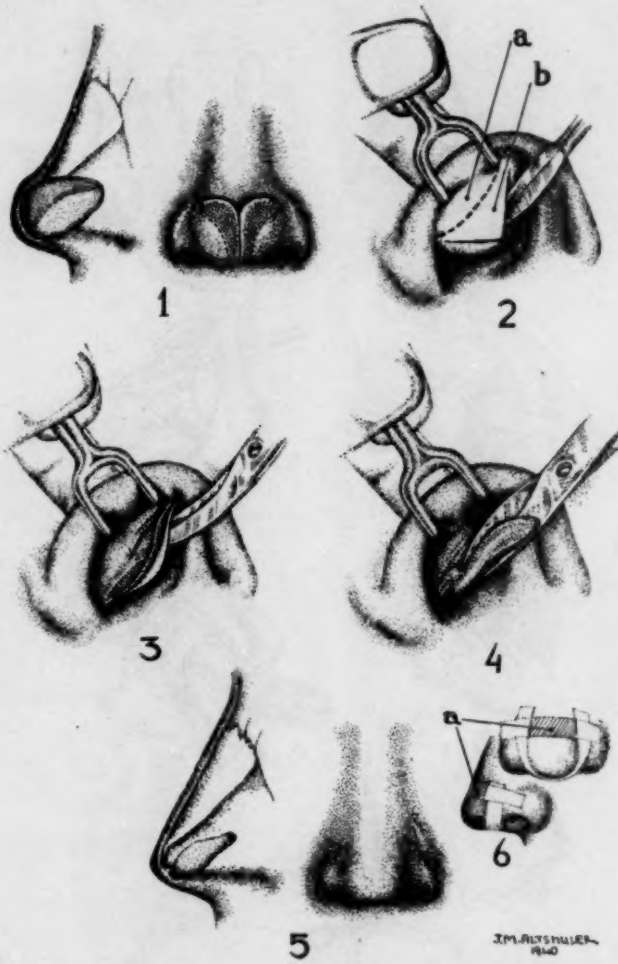

Fig. 4.—An original method for the reconstruction of a bulbous tip: (*1*) the deformity, showing the sections of the alar cartilage to be removed; (*2*) *a* indicates the section of alar cartilage that is left (not removed) and *b* the portion to be removed; (*3* and *4*) the method of removal of the cartilage; (*5*) the amount of cartilage left and the result, and (*6*) the method of postoperative fixation (*a* indicates portions of adhesive which are lined, permitting the skin to shrink).

7. Wolfe, M. M.: Etiology of Saddle Nose: Preliminary Report, Ann. Otol., Rhin. & Laryng. **46**:504 (June) 1937.

porting framework of the nose. The depression may be bony, cartilaginous or a combination of both. Frequently, the cause is septal abscess, faulty submucous resection, syphilis or tuberculosis. I shall exclude from consideration cases in which replacement of the covering

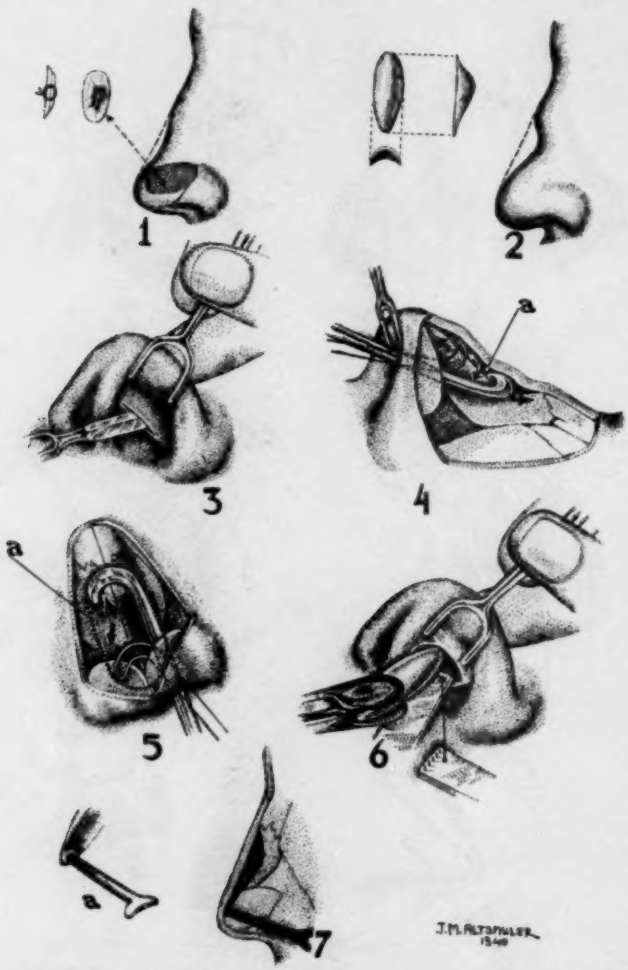

Fig. 5.—Steps in the reconstruction of moderate and severe saddle nose by the use of transplants: (*1*) the deformity corrected by a transplant of alar cartilage; (*2*) a severe deformity and the contour of the transplant necessary for its correction; (*3*) the incision; (*4* and *5*) the formation of a pocket (*a*) for the transplant; (*6*) the introduction of the transplant; (*7*) the transplant in situ, and (*a*) the junction of the transplant with the strut.

or lining of the nose is required. If the saddle is due to a "false hump," which was described earlier in this paper, removal of the hump and

narrowing of the bony arch may suffice without the use of a transplant. In typical saddle nose, the nasal bridge is widened from the trauma, and in order to effect the best cosmetic result it should first be narrowed (fig. 2, *11* to *15*). When the saddle is slight, a small graft of cartilage from the septum or the ear may be utilized or morsels of the previously

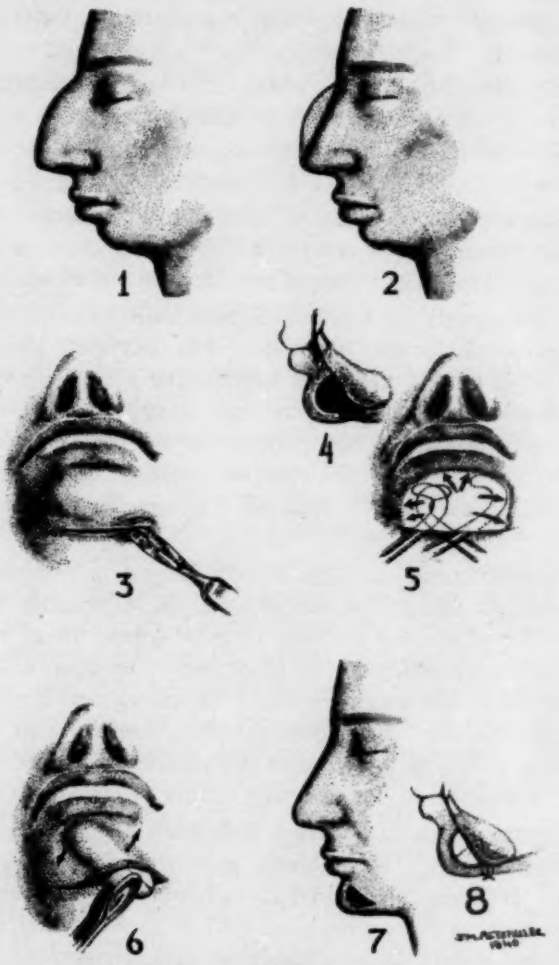

Fig. 6.—Steps in the combined nasal plastic and chin plastic operation: (*1*) a typical deformity; (*2*) the size of the hump removed; (*3*) the submental incision; (*4* and *5*) the formation of a pocket for the placement of the hump in the chin; (*6*) the introduction of the hump, and (*7* and *8*) the results.

removed alar cartilage may be transplanted to the saddle area (fig. 5, *1*). Another corrective method is transposition of the lateral cartilages. If the deformity is a large one, the saddle may be accompanied by a flat-

tening of the nasal tip, especially when it is due to loss of septal cartilage. Therefore, in addition to a transplant for the correction of the saddle deformity, an accurate strut of the same material, extending from the anterior nasal spine to the alar arch, is imperative if the outcome is to be remedial (fig. *7a*).

Transplant Materials: There are several transplant materials available for consideration, each of which has many advocates. I evaluate them as follows:

1. Bone grafts, customarily taken from the iliac crest, require an extensive procedure and are most difficult to shape. They frequently absorb, and there is also the hazard of pressure necrosis of the skin if the graft is improperly shaped or of excessive size.

2. Dermal grafts, consisting of skin of full thickness without epidermis, most recently in vogue, are difficult to gage properly or to fix in place and gradually undergo considerable shrinkage.[8]

3. Ivory infrequently is a favorable transplant material. From a previously prepared plaster cast of the nose it is accurately shaped to fit the deformity. It is readily available, inexpensive and easily sterilized. It is similar chemically to bone but does not absorb. Its use simplifies the procedure and minimizes the operating time; but, like other foreign bodies, ivory is extruded or its removal becomes necessary on account of tissue intolerance or the lack of fixation that almost invariably develops.

4. Autoplastic costal cartilage has the largest number of users,[9] but it is frequently difficult to obtain the large amount of cartilage necessary. Many persons are unwilling to submit to an operation on the costal cartilage, which is the chief source of supply. I have also on occasion found the costal cartilage completely calcified and its use therefore impracticable. Furthermore, when used in large sections cartilage has a tendency to curl and is susceptible to infection, which is a forerunner of absorption and ultimate failure.

5. Refrigerated human cartilage makes the ideal transplant material for all surface defects [10] (this material was originally used by Pierce and O'Connor). It is easily acquired from nonpathologic specimens, especially those from persons who have died from accidental causes. The cartilage is denuded of its perichondrium and refrigerated in a solution of one part aqueous solution of merthiolate to four parts sterile

8. Peer, L. A.: Buried Grafts Used to Repair Depressions in the Brow, Eye Socket, Skull and Nose, J. M. Soc. New Jersey **35**:601 (Oct.) 1938.

9. Peer, L. A.: Cartilage Transplanted Beneath the Skin of the Chest in Man, Arch. Otolaryng. **27**:42 (Jan.) 1938.

10. Pierce, G. W., and O'Connor, G. B.: Refrigerated Cartilage Isografts. Surg., Gynec. & Obst. **67**:796 (Dec.) 1938.

physiologic solution of sodium chloride. It may be stored indefinitely. I change the solution every four weeks, and each time before any of the cartilage is used a culture of the solution is taken. I have so far been unable to get a growth of organisms from this solution. The cartilage can be transplanted regardless of the age, sex or color of the subject. Its use is restricted until it has been refrigerated at least one week. Refrigerated cartilage from the ears of cadavers has also been used successfully by me in total reconstruction of the ear. Thus, it furnishes what has been much sought but never previously found—a satisfactory material for support and shaping of the ear.

Combined Deformity of the Nose and Chin (fig. 6).—A number of persons have both a large hump nose and a small receding underdeveloped chin. The correction of either of the deformities fails to produce the desired esthetic effect. However, the combined correction of rebuilding the chin and reducing the nose is ideal and tends to produce a harmonious profile. After removal, the nasal hump is transplanted into a previously prepared pocket in front of the mandible, through a small submental incision requiring one suture and leaving no noticeable scar.

In some cases the defect of the chin is so marked that it cannot be repaired satisfactorily without the use of a nasal hump of considerable size, which is often absent. Deformity of the chin may exist without a nasal deformity, making the sole requisite the construction of a normal mandibular protrusion. The microgenia is due to faulty development of the mandible. The possessor of such a defect often has an unwarranted complex, for he frequently hears that this deformity indicates weakness of character. When there is an accompanying defect of dental occlusion, this should be corrected by an orthodontist and subsequent plastic repair should be made. This repair may be done with a transplant of any of the materials mentioned for correction of saddle nose. For the reasons given previously, refrigerated human cartilage is the transplant material of choice for this operation.

COMPLICATIONS OF RHINOPLASTIC PROCEDURE

I feel that no paper treating this subject would be complete without mention of the complications, which, fortunately, are seen infrequently. I wish especially to mention a complication which I have encountered several times, to which a thorough perusal of the literature fails to reveal any reference. For lack of a better term, I have called this complication "intranasal keloid," because of its similarity to keloid on any external surface. This condition occurs approximately two to four weeks after nasal repair and is seen along the site of the preliminary intranasal incisions used for approach to the nasal bridge (fig. *2, 4*). It is characterized by induration, thickening and reddish discoloration

along the site of the incisions. This gives the appearance of bilateral overdevelopment of the lateral cartilages (potato nose). Of 4 patients with this complication whom I have seen, 3 responded to mild roentgen therapy over a period of four to six weeks, and the nose gradually became normal. The fourth patient exhibited marked proliferation and remained resistant to roentgen radiation after six months of treatment. Excision of the keloid was followed by rapid recurrence. In cases in which there is a history of keloid, early roentgen therapy immediately following operation seems the minimum requirement in prophylaxis.

There are other complications, as hemorrhage, in which packing and sedation usually suffice. Psychiatric complications are best avoided by having proper psychiatric consultation concerning any patient who appears to be in a borderline state. Many people desiring plastic operations are psychoneurotic and become affected with severe mental disorders after unwarranted plastic intervention. There is also the complication of infection, which occurs infrequently. Simple infections usually localize and are drained through the original intranasal incision, to avoid external scarring. The process of localization takes place between the third and the sixth postoperative day and will subside in a week if adequate drainage is provided early. Constant vigilance, with early adequate drainage for localized infection, is the keynote to success here. Nonlocalizing infections are treated as if no operation had been performed. Strict surgical technic usually prevents infection provided there has been no recent sinusitis or coryza.

SUMMARY

The historical background of rhinoplastic surgery from its inception to the present is reviewed.

The social aspects of plastic surgery are important. Social and professional discrimination gives rise to inferiority complexes in persons having nasal disfigurement.

Rhinoplastic surgery is defined. The scope and problems are of great magnitude. Meticulous technic and careful matching of skin before grafting are essential.

The three periods of nasal reconstruction and the important phases and problems are treated.

Typical nasal deformities may occur singly or in combinations. They are satisfactorily corrected by appropriate procedures, as illustrated by diagrammatic sketches.

An original method is described for the correction of a bulbous nasal tip.

Complications in rhinoplastic procedure occur infrequently and demand judicious treatment. The novel complication of "intranasal keloid" is reported.

MUCOCELE OF THE FRONTAL SINUS

REPORT OF A CASE

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Although mucocele of the frontal sinus was not dealt with as a problem in therapeutics in authoritative medical literature earlier than the eighteenth century, this pathologic condition must have existed in ancient times. An interesting bit of writing indicating this is the description by Boenninghaus [1] of a skull discovered by him among the thousands ornately arranged on the walls of the famous chapel at Tscherbeney, built entirely of human bones. Boenninghaus made a careful examination of this skull. He found the bone structure large and strong, with the teeth for the most part absent; the skull appeared to be that of a person of middle age. The cranial capsule was perfectly preserved. A large part of the anterior wall of the frontal sinus was absent. In the cerebral wall of the left frontal sinus was a roundish defect, approximately 1 cm. in diameter. From these and other observations Boenninghaus concluded that the person had suffered from mucocele of the frontal sinus.

The first physician to collect records of mucocele of the frontal sinuses was the French pathologist Berthon.[2] In 1880 he published his "Essai sur les abscès et hydropisies des sinus frontaux," in which he recorded cases of other physicians. Some of the most interesting and valuable outlines of cases in Berthon's review are taken from a report of Dezeimeris, published in 1839 in the journal *L'Expérience*. The earliest of these case reports outlined by Dezeimeris is that of Henry Albert Nicolai, which appeared in a medical journal published in Strasbourg, France, in 1725. The case report is interesting as a historical contribution to the therapeutics of mucocele. The patient was a soldier who suffered from extremely painful headaches. No physician seemed able to find a remedy. These headaches had begun three years previously, at which time the soldier had suffered a fracture of the skull from a sword cut. The treatment prescribed was "powder, emulsion, and external remedies." The second day after beginning treatment, the patient felt an "explosion" in his head. This was

1. Boenninghaus, G.: Zur Kenntnis der traumatischen Mukocele des Sinus frontalis, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **3**:116-124, 1909; Jahresb. d. schles. Gesellsch. f. vaterl. Kult. (Med. Sect.) **76**:211-212, 1898-1899.

2. Berthon, E.: Essai sur les abcès et hydropisies des sinus frontaux, Thesis, Paris, no. 263, 1880.

followed by a thick purulent nasal discharge, streaked with blood, which lasted for a day and resulted in complete cessation of pain.

A second early case report of interest is that of Johannes Ludwig Welge,[3] published in his dissertation "de morbis sinuum frontalium." He described a case of "suppuration in the frontal sinus," with considerable dilatation of this cavity, occasioning, by pressure exerted on the skull, a comatose state, and considerable tumefaction of the visage. The condition resulted from "old venereal disease." During the patient's hospitalization, a splinter of bone was removed from the orbital cavity and the ethmoid sinus. Mercuric chloride, followed by Plummer's powder, was administered, "with mercurial unctions around the eye." The frontal and maxillary sinuses and the nasal fossae were found to be merged into a single cavity. The patient refused further treatment and left the hospital.

In 1805 Doerner [4] described the case of a man struck in the left eye by a falling saber. The trauma resulted in a large tumor on the upper eyelid. In this report, one notes advances in treatment, including thorough lavage, careful exploration of the sinus and trepanation of the bone. Two years later Horn published in the *Archives of Horn* the case of a 16 year old girl who suffered from headache, pain in the root of the nose, dizziness, faulty vision and a sensation of fluctuation above the nose. The condition was recognized as inflammation of the frontal sinus and treated by "friction with volatile liniment, administration of a sternutatory powder, fumigation with steam, and nasal injections. The flow of pus thus started was treated with lavage, and, after the patient sneezed violently, there was a profuse nasal discharge of fetid, tenacious, mucopurulent material, evacuation of which was followed by great relief and complete recovery."

The case of Professor Langenbeck,[5] reported in 1818 in "Neue Bibliothek für die Chirurgie und Ophthalmologie," is considered by some authorities to be the first authoritative report of a case of mucocele of the frontal sinus. At any rate, Warlomont [6] considered it of sufficient importance to be included, in the translation, in his textbook, "Le traite pratique des maladies de l'oeil de Mackenzie."

Other early case reports are of interest particularly from the historical point of view.[7] A perusal of these, all cited by Berthon,[2] will

3. Welge, J. L.: De morbis sinuum frontalium, Göttinger, J. C. Dieterich, 1786.

4. Doerner, cited by Berthon.[2]

5. Langenbeck, C. J. M.: Neue Bibliothek für die Chirurgie und Ophthalmologie, Hanover, Hahn, 1820, vol. 2, p. 365.

6. Warlomont: Le traite pratique des maladies de l'oeil de Mackenzie, ed. 4, vol. 1, p. 77.

7. Tott, C. A.: Entzündung der Stirnhöhlen mit gleichzeitiger Taubheit und Amblyopie, J. f. Chir. u. Augenh. **11**:662, 1828. Wisbach: Mag. d. ausl. Lit. d. ges. Heilk. **19**:512, 1830. Riberi, A.: Esostosi entrorbitale guarita coll'estirpazione,

(Footnote continued on next page)

give the reader a good idea of the handling of mucocele of the frontal sinus by physicians up to the latter part of the nineteenth century. In the twentieth century, medical practice has advanced by leaps and bounds far beyond the early and often crude concept. Nevertheless, all that is known and all that is done today is based, step by step, on the foundation laid by those hard working poorly equipped but patient and determined pioneers in the art of healing.

DEFINITION

Stedman, in his medical dictionary, defined a mucocele simply as a cyst containing mucus. McKenzie [7a] wrote: "The modern opinion is that a mucocele is a mucus cyst (and it in turn a form of adenoma) which, undergoing active enlargement, leads to a distention of the bony walls of the sinus and their slow atrophy."

Raoult,[8] Axenfeld,[9] Pereira and Adrogué,[10] Rollet [11] and other authorities have agreed that the growth of mucocele of the frontal sinus results in a rounded painless smooth swelling in the roof or inner wall of the orbit. Usually making its appearance just above the inner canthus, the swelling may involve the entire upper lid or the forehead or most of the nose. On the other hand, a mucocele of the lower ethmoid region extends downward into the nose, appearing as a large middle turbinate. As a rule, the patient complains of visual disturbance or of a dull heavy headache or of both.

On examination, no inflammatory changes are noted over the swelling, and no tenderness unless a secondary infection has taken place. Crepitus may be present when the bone becomes thin and cracks. After absorption of bone has taken place, the edges of the bone may be felt. The eyeball may be displaced outward, downward and forward. Yet

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Gior. d. sc. med. di Torino **3**:447, 1830; cited, J. de chir. (Malgaigne) **4**:24, 1845. Bouyer, M.: Essai sur la pathologie des sinus frontaux, Thesis, Paris, no. 121, 1859, p. 49. Demarquay: Abscès des sinus frontaux, Gaz. d. hôp., Feb. 27, 1866. Richet, cited by Sautereau, F.: Étude sur les tumeurs de la glande lacrymale, Thesis, Paris, no. 10, 1870, p. 68. Cruveilhier: Abscès des sinus frontaux survenu sous l'influence de la syphilis, Bull. Soc. anat., 1880, p. 167.

7a. McKenzie, D.: Diseases of the Throat, Nose and Ear, ed. 2, St. Louis, C. V. Mosby Company, 1928, vol. 1, p. 304.

8. Raoult, A.: Mucocèle du sinus frontal, Bull. d'oto-rhino-laryng. **19**:1-131 1921.

9. Axenfeld, T.: Latentes Osteum und Mucocele des Sinus frontalis, Klin. Monatsbl. f. Augenh. **42**:229-232, 1904.

10. Pereira, R. F., and Adrogué, E.: Sobre un caso de mucocele del sinus frontal, Rev. de especialid. **4**:255-265, 1929.

11. Rollet: Mucocèle du sinus frontal, Bull. Soc. de chir. de Lyon **10**:131-133, 1907.

the changes are wrought so slowly that the occurrence of diplopia is comparatively infrequent. The mass may feel soft and elastic on palpation or may be found to fluctuate. The appearance of the nares is not usually suggestive, yet at times a bulging in the region of the anterior third of the middle turbinate is indicative.

ETIOLOGY

Many and varied opinions have been expressed in regard to the causative factors of mucocele. According to most authorities, trauma plays the most important part. Other than trauma I have been unable to find any real cause which is worthy of emphasis. The following is a summary of probable and possible etiologic factors presented or suggested in the literature of mucocele of the frontal sinuses.

Trauma.—As has been stated, trauma seems to be the most frequent cause of mucocele. This may be illustrated by the following cases:

Airoldi [12] had a patient who five years previously had accidentally struck her forehead and nose against the corner of a window blind. No pathologic condition appeared for three years. Berthon [2] (as I have mentioned) reported the case of a soldier whose skull was fractured by a blow from a sword. Lieutaud [13] had a patient whose postmortem record showed that he had been hit two years previously with a piece of iron on the superior border of the orbit. Doerner's [4] case of trauma caused by a saber falling on one eye has been mentioned. Langenbeck [5] cited the case of a youth who nine years previously had struck his temple on the corner of a table; a mass had begun to form soon afterward. Boenninghaus [1] described several cases in which mucocele of the frontal sinus was preceded for variable periods by some sort of trauma. One of de Vincentiis' [14] patients had been kicked by a horse in the region of the right eye thirteen years previously and for two years before presentation for treatment had suffered from severe headaches, for the most part on the right side. A woman of 60, treated by di Santo,[15] had had for approximately twenty years intermittent pains in the head accompanied by catarrh. She had been accidentally struck by her husband's elbow over the right eyebrow. One

12. Airoldi, A.: Sopra un caso di mucocele fronto-etmoidale, Gazz. med. lomb. **66**:268-273, 1907.

13. Lieutaud, cited by Berthon.[2]

14. de Vincentiis, C.: Mucocele frontale ed innesto di un lembo di cornea in un occhio affetto da piccola ernia dell'iride, Lavori d. clin. ocul. d. r. Univ. di Napoli **3**:330-331, 1891-1893.

15. di Santo, C.: Mucocele suppurato dei seni frontali con mucocele del seno etmoidale destro, Prog. oftal. **2**:40-50, 1906-1907.

of Gross's [16] patients was a woman of 45 who had received a blow on the forehead when she was 10. Gaus [17] described the case of a man of 50, struck on the left eyebrow by a piece of sharpnel twelve years previous to presentation. From that time he had had recurrent abscesses and intermittent protrusion of the frontal region. Hamaoua,[18] in his valuable review of outstanding cases of mucocele other than his own, cited Angelo Casali's case, in which a mucocele was ascribed to frontal traumatism. Lawson [19] described the case of a man of 58 with a large frontal tumor, whose history recorded that at the age of 4 he had been kicked in the eyebrow. Among other authorities who described cases in which mucocele was either caused or affected by trauma are Acomb,[20] Megret,[21] Rollet,[11] Teillais,[22] Benoit,[23] Kriebel,[24] dall'Olio,[25] Sacco,[26] van den Wildenberg,[27] Berger,[28] Briani,[29] Higgins,[30] Kuhnt,[31] Michail,[32] Padula,[33] Raoult,[8] Fox [34] and Garretson.[35]

16. Gross: Un cas de mucocèle de la région frontale, Mém. Soc. de méd. de Nancy (1887-1888), 1889, p. xxxi.

17. Gaus, W.: Ueber einem Fall von Mucocele der Stirnhöhle nach Schuszverletzung und seine Behandlung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:224-227, 1935.

18. Hamaoua, E. J.: Des mucocèles du sinus frontal, Thesis, Paris, no. 436, 1911.

19. Lawson, G.: On the Treatment of Distension of the Frontal Sinus from Pent-Up Secretions of Pus, Practitioner **5**:8-12, 1870.

20. Acomb, J.: Mucocele of Frontal Sinus, Brit. M. J. **1**:344-345, 1922.

21. Megret, R.: De la mucocèle fronto-ethmoïdal, Thesis, Paris, no. 476, 1920.

22. Teillais: Kyste du sinus frontal, J. de méd. de l'ouest **15**:16-22, 1881.

23. Benoit, A.: Mucocèle du sinus frontal, Arch. internat. de laryng. **5**:316-320, 1926.

24. Kriebel, A.: Traumatische Mukozelen der Nebenhöhlen der Nase, Ztschr. f. Laryng., Rhin. **19**:34-42, 1930.

25. Dall'Olio, C.: Di un raro caso di mucocele fronto-etmoidale bilaterale, Oto-rino-laring. ital. **3**:419-427, 1933.

26. Sacco, A.: Mucocele o quista epitelio glandular del seno frontal, Bol. y trab. de la Soc. de cir. de Buenos Aires **7**:241-248, 1923.

27. van den Wildenberg, L.: Volumineux mucocèle ethmoïdo-frontal, Arch. internat. de laryng. **34**:147, 1912.

28. Berger, A. P. G.: Fünf Fälle von Erweiterung der Stirnhöhlen durch Flüssigkeitsansammlung, Inaug. Dissert., Kiel, Schmidt u. Klaunig, 1887.

29. Briani, A. A.: Mucocele del seno frontale, Gior. med. d. Alto Adige **3**:509-515, 1931.

30. Higgins, C.: Three Cases of Distension of the Frontal Sinus, Guy's Hosp. Rep. **1**:43-47, 1894; Distension of Frontal Sinus, ibid. **25**:27-40, 1881.

31. Kuhnt, H.: Mukokele der Stirnhöhle und des Siebbeinlabyrinths mit plötzlichem hochgradigen Exophthalmus, Ztschr. f. Augenh. **33**:64-75, 1915.

32. Michail, D.: Fold Formation of Retina Caused by Fronto-Orbital Mucocele, Cluj. med. **13**:290-294, 1932; Ann. d'ocul. **171**:126-128, 1934.

33. Padula, F.: Mucocele dei seni frontale con usura dell'osso, Riv. ven. di sc. med. **10**:70-74, 1889.

(Footnotes continued on next page)

Respiratory Disorders.—A considerable number of cases are on record in which, according to the reports, mucoceles were preceded more or less recently by various respiratory infections. Buller [36] treated a woman of 67 who for forty years had had catarrh and polypi. Hubert [37] described the case of a patient who had had grip followed by frontal pain; then tumefaction had appeared and progressed rapidly. Beyer's [38] patient, a woman of 52, had had catarrh and a pathologic condition of the frontal sinus for ten years. Barwell [39] treated for mucocele a boy of 12 who had been suffering for two months from a severe head cold. One of Higgins' [30] patients was a youth of 22 who for years had had chronic catarrh accompanied by headache. Portmann and Teulières [40] described the case of a patient who a year previously had suffered frontal pain together with a succession of colds. In Bonnet's [41] case, a report of which was published in 1935, giant mucocele was preceded by "cold in the head" accompanied with an extremely abundant discharge from the nose. When this stopped suddenly after a few days, the eyelids became permanently swollen. In several weeks a tumefaction appeared.

Canuyt's [42] patient had had frequent colds and had undergone an operation for nasal polypus. A case described by Francois [43] was that of a patient who had a tuberculous lesion of the lung and had had an abscess of a right upper tooth eight years previously, dental caries (which had necessitated extraction of all the teeth), benign gastric and intestinal disturbances, parenchymatous goiter (still present) and for ten years chronic coryza, with persistent blockage of the nose by a purulent discharge. This chronic rhinitis was frequently accompanied by acute phases of "colds in the brain," with abundant mucopurulent discharge, frontal pains and sensations of heaviness and numbness in the region of the right frontal sinus. Geschelin [44] described the case

34. Fox, M. C.: Mucocele of Frontal Sinus, Northwest Med. **25**:30-33, 1926.

35. Garretson, W. T.: Mucocele of Frontal Sinus, Laryngoscope **37**:350-354, 1927.

36. Buller, F.: Mucocele of Frontal Sinus, Am. J. Ophth. **1**:33-37, 1884-1885.

37. Hubert, C.: Cas de mucocèle frontal suppurée ouverte dans les fosses nasales, Oto-rhino-laryng. internat. **16**:120-121, 1932.

38. Beyer, T. E.: Pyocele of Frontal Sinuses, Laryngoscope **42**:715-719, 1932.

39. Barwell, E.: Suppurating Mucocele of Frontal Sinus in a Boy Aged Twelve, Proc. Roy. Soc. Med. (Sect. Laryng.) **6**:112, 1912-1913.

40. Portmann, G., and Teulières, M.: Mucocèle et sinusite frontale, J. de. méd. de Bordeaux **54**:379-381, 1924.

41. Bonnet, P.: Mucocèle géante du sinus frontal, Bull. Soc. d'opht. de Paris, 1935, no. 2, pp. 728-732.

42. Canuyt, G.: Sur un cas de mucocèle fronto-ethmoïdale gauche extériorisée, Bull. Soc. d'opht. de Paris, 1927, pp. 293-296.

43. Francois, J.: Ostéopériositite supéro-interne symptomatique d'une mucocèle infectée du sinus frontal, Bull. Soc. belge d'opht., 1931, no. 63, pp. 83-97.

44. Geschelin, A. I.: Pyo-Mucocele fronto-ethmoidalis, Acta oto-laryng. **21**: 377-385, 1934.

of a young woman of 23 who had had influenza when she was 12. He stated the belief that in this connection purulent frontal sinusitis had developed. This opened a channel spontaneously under the skin and was drained without the source of a purulent accumulation having been sought. The affected sinuses were shut off from the nasal cavity, and a many-chambered ethmofrontal cyst was formed, most likely due to scar. The mucosa again threw off profuse secretion. This caused the external wall of the cyst to dip gradually into the ocular cavity. Almost ten years elapsed before the patient became conscious of the tumor in the angle of the eye, which appeared and then disappeared for six months. About this time a fistula developed, and later an enlargement of the avenue of communication between the frontal sinus and the ocular cavity. As the communication increased, the tumor acquired a constant character.

One of Berger's [28] patients (case 5) had had diphtheria three years before presentation for treatment for mucocele. My own patient with mucocele, whose case is to be described later, had had an attack of acute bronchitis some three weeks before he was examined.

The reader, if interested in further data on this aspect of the etiology of mucocele, is referred to Engelmann's [45] valuable treatise on catarrh of the frontal sinus. Among other authorities who have dated the advent of mucocele from coryza, influenza, nasal blockage and similar conditions are Labit,[46] Herrnheiser,[47] Zanni,[48] Dehogues and Martínez,[49] Kuster,[50] Peyrot,[51] Paunz,[52] Dabney [53] and Valude.[54]

Venereal Disease.—Morini,[55] in an interesting study of mucocele of the frontal sinus, offered the hypothesis that the condition may be

45. Engelmann, V.: Der Stirnhöhlenkatarrh, Arch. f. Laryng. u. Rhin. **1**:291-359, 1893-1894.

46. Labit, G.: Sur un cas de catarrhe purulent des sinus frontaux, Ann. de la Polyclin. de Bordeaux **1**:13-15, 1889.

47. Herrnheiser, G.: Der Röntgenbefund bei der Mucocele oder Pyocele der Stirnhöhle und der Siebbeinzellen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:319-334, 1926.

48. Zanni, G.: Sulle complicazione orbitarie di origine sinusale, Riv. oto-neuro-oftal. **9**:97-108, 1932.

49. Dehogues, J. L., and Martínez, E.: Quiste por retención del seno frontal derecho, Arch. de la Polyclín., Habana **1**:134-141, 1896.

50. Kuster, E.: Hydrops sinus frontalis, Med. chir. Centralbl. **17**:435-436, 1882.

51. Peyrot: Des hydropisies et des abcès des sinus frontaux, Paris méd. **6**:385-387, 1881.

52. Paunz, M.: Beiträge zur Mukokele der Stirn- und Siebbeinhöhlen, Cong. internat. de méd., Compt. rend. (Sect. ophth.) **16**:387-389, 1909.

53. Dabney, V.: Mucocele of Nasal Accessory Sinuses, New York M. J. **114**: 619-623, 1921.

54. Valude, E.: De la mucocèle du sinus frontal, Ann. d'ocul. **122**:424-433, 1899.

55. Morini, E.: Sopra un caso di mucocele del seno frontale destro, Ann. di ottal. **31**:739-742, 1902.

caused by a syphilitic gumma. He described a case in which a gumma was localized in the orbital wall of the frontal sinus, causing by its enlargement continual irritation of the mucosa. Then the osseous floor, eroded by the gumma, allowed the mucocele to enter the orbit. This gave rise to violent circulatory phenomena. I have already referred to Welge's [3] early case, in which he attributed the patient's condition to "old venereal disease."

Infectious Diseases.—Scarlet fever and measles have been credited by some authorities with creating conditions favorable to the development of mucoceles. Both Ferreri [56] and Nietsch [57] described mucoceles, the development of which was preceded by scarlet fever. Benjamins [58] said that he considered measles a probable contributing factor. Higgins [30] had a patient, a man of 32, who for eight months had had a tumor of the frontal region. Twenty-five years previously he had had an abscess on the inner canthus after an attack of scarlet fever. Eighteen years before, he had suffered a blow on the head, which was followed by the expulsion of necrotic bone.

Miscellaneous.—Sunstroke, suffered several months before inception of the tumor and causing intense redness of the forehead, was mentioned by Boel (see Crebessac [59]) as a possible causative factor. Zambrini and Casteran [60] described a case in which sunstroke followed after two weeks by a blow on the head preceded the appearance of a mucocele. Demarquay, cited by Berthon,[2] referred to a patient who had good health until the age of 18, then suffered an attack of generalized articular rheumatism. Some months later a series of attacks of supraorbital neuralgia began. Scialdoni [61] treated for mucocele a woman of 64, who at 22 had suffered about a month from headache of a "rheumatic nature." Sobol [62] mentioned a patient whose illness began with erysipelas of the left side of the face three years before presentation for treatment. Lawson [19] had a similar case, that of a woman of 21 who had had a tumor for six years. The trouble had begun fifteen years previously, after an attack of erysipelas.

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56. Ferreri, G.: Muco-pneumatocèle géante bilatérale des sinus frontaux, Arch. internat. de laryng. **28**:973-975, 1922.

57. Nietsch: Ueber einem Fall von Hydrops des Sinus frontalis dexter, Prag. med. Wchnschr. **20**:156-157, 1895.

58. Benjamins, C. E.: Aeussere oder innere Operation bei Mukozele der Stirnhöhle, Ztschr. f. Laryng., Rhin. **16**:447-452, 1928.

59. Crebessac, P.: Contribution à l'étude des mucocèles du sinus frontal, Thesis, Paris, 1924.

60. Zambrini, A. R., and Casteran, E.: Secuestro y mucocele supurado consecutivo a fractura del frontal, Rev. Asoc. méd. argent. **46**:788-789, 1932.

61. Scialdoni, A.: Storia clinica di un caso d'idropisia del seno frontale destro, Gior. internaz. d. sc. med. **5**:888-895, 1883.

62. Sobol, I.: Sur l'étiologie et le diagnostic des mucocèles des sinus du nez. Oto-rhino-laryng. internat. **17**:730-741, 1933.

For further discussion of etiologic factors, the reader is referred to the valuable contribution of Reverchon and Worms [63] on the genesis of mucocele.

SIGNS AND SYMPTOMS

Diagnosis of a true mucocele of the frontal sinus is difficult, because the condition is characterized by so great a number and variety of signs and symptoms. Nevertheless, some of these are fairly constant. The more common signs and symptoms have been previously discussed. Less common signs are sluggish pupil reaction, inflammation or congestion of the conjunctiva, impairment of the oculopalpebral sulcus, epiphora, diplopia and edema. More rarely found are myopia, strabismus, dermatitis, anisocoria, photophobia, astigmatism, ulceration of the cornea, papillitis, opacities in the crystalline lens and lack of light perception. Strange to say, in a few cases (usually those in which the lesion is not far advanced) involvement of the eyes has not been evident.

It may be of interest to cite a few outstanding and unusual findings. Forster [64] had a patient with such marked displacement and proptosis that the eyelids could not be closed. This patient, a man of 30, was blind. Wong's [65] case was also complicated by blindness and marked by extreme swelling and displacement downward and outward, accompanied by severe continuous headaches. Snell [66] reported a case of mucocele in which the eye was completely atrophied. In Michail's [32] case, a giant mucocele had produced corrugation of the retina. Zentner [67] had a patient who presented compression and wrinkling of the bulb.

Further references on the diagnosis and symptoms of mucocele of the frontal sinus are the articles of Arcelin,[68] Malan,[69] Sobol,[62] Tewfik [70] and Herrnheiser.[47]

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63. Reverchon, L., and Worms, G.: Mucocèle fronto-ethmoïdale. Pathogénie, traitement, Arch. internat. de laryng. **28**:682-691, 1922.

64. Forster, H. V.: Mucocele of the Left Frontal Sinus. Proc. Roy. Soc. Med. (Sect. Laryng.) **16**:18, 1922.

65. Wong, A. S.: Mucocele of the Left Frontal Sinus and Ethmoidal Cells, China M. J. **45**:991-993, 1931.

66. Snell, J. A.: Unusual Cyst of Frontal Sinus, China M. J. **44**:472-474, 1930.

67. Zentner, P.: Ungewöhnliche Mukozele der Stirnhöhle mit Kompression und Faltung des Bulbus, Klin. Monatsbl. f. Augenh. **80**:508-512, 1928.

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PATHOLOGY

The pathologic appearance of a mucocele of the frontal sinus is fairly constant, varying only with the size of the growth and the pressure exerted by it. The tissue changes are too well known to require description or interpretation. Various types of pathologic development can be found in the cases herein reported; therefore, this phase of the disease requires no special discussion.

This paper deals mainly with mucocele of the frontal sinus as a primary, not a secondary, condition.

Unilateral Mucocele.—As an aid to diagnosis as well as in an endeavor to add to the general knowledge of the pathologic changes attending a true mucocele of the frontal sinus, I shall cite more detailed outlines of 6 illustrative cases.

1. Raoult [8] reported the case of a boy of 13 who at the age of 7 had been struck by a bottle at the level of the root of the nose. Since that time he had had a rounded projection above the internal angle of the left eye. This projection had remained stationary until approximately three months before presentation for treatment. For some time the patient had suffered from a discharge of mucus through the left naris. When dissection and incision of the cyst were made, a quantity of thick, very viscous, slightly yellow pus issued forth. A second cystic pocket was discovered at the level of the internal wall of the orbit. This cyst was separated from the first cyst by an osseous bridge, which was removed with gouges. When the entire anterior wall of the mucocele had been removed and the cavity cleaned, it was found that the cavity was formed by four spaces, the two larger being separated partly by osseous lamellae, which were removed with a forceps. At the margin of the external wall, formed largely of fibrous tissues, there were again some very thin osseous lamellae separating the mucocele from the orbit. These lamellae were dissected and removed. The mucosa which lined the cystic cavity was reddened and "seemed very thin." This cavity, somewhat irregular, was large enough to contain a "large nut." The condition was diagnosed as true mucocele.

2. Axenfeld [9] described the case of a man of 40 who had had displacement of the left eye for several years, with a protrusion under the roof of the ocular cavity. There had been no pain. At the time he was seen, the tumor was softer than at its inception. There was no recollection of trauma or any other inducing factor. Typical Krönlein's resection of the external orbital wall was done, revealing "true mucocele of the frontal sinus." In the course of the operation it was discovered that the frontal sinus was distended and practically full of nodulated bone tumor. When the attenuated anterior wall had been removed, together with the tumor, it was found that the osteoma was inserted in a septum dividing the original frontal sinus into two parts. Toward the nose there was a small, intact frontal sinus and toward the temple there was a large cavity, in which the osteoma had developed. When the osteoma into the septum had been severed with a chisel, the tumor detached itself on all sides from the walls of the sinus, to which it had been obviously only adjacent, not attached. The diagnosis was true mucocele with typical content discharged when operative rupture occurred. The case was atypical and of particular interest because of the septum in the sinus and the bony growth.

3. One of Padula's [83] patients was a woman of 40 who was suffering from insomnia and pain from a tumefaction of the forehead. This had at one time been punctured, with temporary relief. At the time the woman was examined, the tumefaction had reappeared and grown to the size of a pigeon's egg. It was raised almost 2 cm. from the forehead and extended from the root of the nose to the middle of the brow. The consistency was that of bone. There was almost a complete substitution of the nasal pyramid by a triangular foramen, with the vertex above. This aperture measured 3.5 cm. in its greatest width, extending from the upper limit of the lip to the root of the nose. There was a history of severe trauma fifteen years before, followed by loss of almost all of the nasal pyramid. Examination showed loss of substance of a good half of the nasal bones; the cartilaginous septum had disappeared, and only a small portion of the osseous septum remained. The margin of the abnormal foramen described above was red, tumefied and covered here and there with crusts. In this vicinity the redness and swelling extended into the skin up to the root of the nose and to the internal angles of the eyes. The nasal cavity (only anteriorly, because of destruction of the septum) was lined with mucosa which had livid spots covered by a yellowish crust with a fetid odor. It was not possible to find any opening communicating with the frontal sinuses. After the fluid had been extracted, the finger applied over the skin of the glabella could palpate in the frontal bone an irregular, circular depression about 1 cm. deep and with a diameter of about 2.5 cm.; the border, resistant and sharp, was slightly elevated from the surface of the forehead. It was obvious that the anterior wall of the frontal sinuses had been pushed forward and that it was eroded in the middle. The diagnosis was mucocele of the frontal sinuses.

4. Pereira and Adrogué [10] described the case of a boy of 14 who up to his fifth year had "suffered with convulsions." Palpation of the left side of the nose at the level of the superior medial angle of the orbit revealed a continuation of the frontal wall, which had been lowered about 1 cm. Palpation of the more external part of this wall revealed a lobulated and resistant tumor, in some parts eburneous, of definite limits and the size of a hen's egg. There was no pain and no pulsation. The left eye presented marked exophthalmos, accompanied by displacement of the globe downward. The frontal sinuses were transparent on transillumination. The nasal fossae showed no suppuration. Roentgen examination revealed considerable dilatation of the sinus toward the internal wall of the left orbit. The left superior orbital arch was displaced slightly downward. When the anterior wall of the frontal sinus was opened, a large quantity of mucous liquid issued forth. The condition was designated as mucocele of the frontal sinus.

5. Rollet [11] reported the case of a woman of 25 who for ten years had had a small, slowly enlarging tumor at the superior internal angle of the orbit. There was no inconvenience, no pain, neither deviation of the eyeball nor diplopia, and the fundi oculorum were normal. Ten years previously the patient, without losing consciousness, had been struck by a cow's horn at the level of the superior internal angle of the right orbit. Shortly thereafter a small tumor had made its appearance. When the patient presented herself for examination, the tumor had attained the size of a small nut. The skin was smooth, of a normal color and without adhesions. Palpation revealed a cystic content. A rather large hyperostosis was felt at the internal and superior angle of the orbit. The tumor was nonpulsatile and nonreducible and did not move or change size. Examination of the nasal fossae revealed the presence of numerous polyps in the right nasal fossa. Transillumination of the sinuses showed that the right frontal sinus was cloudy. This author emphasized the fact that orbital hyperostosis is a valuable sign of frontal mucocele, whether or not of traumatic origin.

6. One of Valude's [54] patients was a woman of 49 who came to him with a tumefaction the size of a small nut at the right superior internal part of the orbit below the arch. The swelling yielded slightly to pressure, and a rounded deep orifice could be recognized, which possibly gave access to the frontal sinus. It was sensitive, even painful on pressure, but no spontaneous pain was present. The eyes were widely spaced, because of hypertrophy of the nasal skeleton, and the frontal region projected prominently, especially in the medial region. The base of the nose was enlarged. There was no history of "scrofula," syphilis or traumatism, —"just a recent head cold" followed by nasal obstruction. Exophthalmos of the right eye, with diplopia, and slight papillary edema were noted. The vision was not impaired. Exploratory puncture brought forth a transparent fluid, clear rose-yellow, colloidal, coagulating almost immediately, to separate later into two parts. After the excision of a cyst, the mucosa of the sinus was curetted. The tissue thus removed was found to be very much vascularized; all the vessels were filled with blood, and the walls of the largest were considerably thickened. There was also alteration of the epithelium; some regions were normal, while others showed flattening, cornification and stratification. No other disease was apparent. The diagnosis was mucocele of the frontal sinus.

Bilateral Mucocele.—Bilateral mucocele is a rare condition. Gerber [71] selected 178 cases of mucocele and found among them only 22 in which the condition was bilateral. Both Benjamins [58] and Airoldi [12] described cases in which the growths were of bilateral type. Ferreri [56] operated successfully on a giant bilateral mucopneumatocele. In dall'Olio's [25] case the floor of the frontal sinus showed extensive erosion on both the right and the left side, with bilateral diffuse osteitis, of the floor, and there was destruction of the peri-infundibular anterior ethmoid cells. The cause was probably trauma. In de Vincentiis' [72] case bilateral mucocele was accompanied by spontaneously pulsating exophthalmos. Van Voorthuysen [73] described a mucocele of both of the frontal and ethmoid sinuses.

Giant Mucocele.—Among the authorities describing cases of giant mucocele are Airoldi,[12] Boenninghaus,[1] Bonnet,[41] Ferreri,[56] Cange,[74] Triossi,[75] Valerio,[76] van den Wildenberg,[27] Zanni,[48] Zentner [67] and Dowman.[77]

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71. Gerber, P. H.: Die Komplikationen der Stirnhöhlenentzündungen, Berlin, S. Karger, 1909.

72. de Vincentiis, C.: Di un mucocele d'oppio dei seni frontali e di un esottalmo pulsante spontaneo, Lavori d. clin. d. r. Univ. de Napoli **3**:332-335, 1891-1893.

73. van Voorthuysen, D. G. W.: Case of Bilateral Fronto-Ethmoidal Mucocele, Geneesk. tijdschr. v. Nederl.-Indië **74**:589-597, 1934.

74. Cange, A.: Les mucocèles géantes du sinus frontal à évolution orbitaire, Arch. d'opht. **39**:193-229, 1922.

75. Triossi, S.: Di un caso di mucocele gigante del seno frontale, Riv. oto-neuro-oftal. **8**:63-76, 1931.

76. Valerio, M.: Considerazioni tratte dall' esame di un caso di mucocele gigante del seno frontale, Riv. oto-neuro-oftal. **12**:585-595, 1935.

77. Dowman, C. E.: Giant Mucocele of the Frontal Sinus, J. A. M. A. **81**: 1014-1016 (Sept. 22) 1923.

Involvement of the Brain and of the Dura.—Cases of mucocele of the frontal sinuses are occasionally observed in which the pathologic condition is so deep and so extensive that surgical intervention results in exposure of the dura mater. In almost all such cases, as recorded in the literature, the close proximity of the mucocele has seemed to have little or no permanent effect on the dura or the cerebrum. Cases in which there is exposure of the dura and of the brain are so valuable clinically, not only because of the unusual interest of the pathologic conditions involved but also because of the extreme difficulty of handling, that I have considered it justifiable to outline briefly all those of outstanding significance which I have been able to find in the literature of mucocele of the frontal sinus. The literature reveals no case in which the cerebrum was exposed by an uncomplicated mucocele. However, it will be noted in the case report which I shall give further on that the cerebrum was exposed because of the destruction of the dura, the pia and the arachnoid.

1. Cange,[74] of Algiers, Algeria, reported a case of true mucocele in which, after removal of the existing tumor, the dura mater could be seen through an osseous dehiscence the size of a lentil, which occupied the center of the new orbital vault. The dura was intact. "Operative result perfect."

2. Petterino Patriarca and Rosello [78] reported a mucocele with coexisting osteoma, which had invaded the entire posterior prolongation of the sinus. The intersinusal septum was eroded, and part of the right frontal sinus had been invaded by the tumor. When removal of the osteoma was attempted, it was noted that on the posterior wall of the sinus there was a small zone about 0.5 cm. square, from which it was impossible to detach the tumor. This was obviously the point of origin. Here a small area was extirpated, a procedure which brought into view the dura. This was immediately touched with iodine. The patient was completely relieved and made a good recovery.

3. Schneirer [79] presented a case of fluctuating tumor, operative exposure of which showed it to be of an elastic consistency. The mucosa was very thin and translucent. The posterior wall was also attenuated, and at one place near the base there was an area of softening. Here the dura showed through. The septum narium protruded, saddle shaped; the lower angle of the frontal sinus had replaced the anterior ethmoid cell. The patient recovered.

4. Paunz [52] described an interesting case of mucocele of the frontal and ethmoid sinuses, in which impairment of the dura occurred. When the bone from the posterior wall of the frontal sinus was removed by operative procedure, the dura was exposed and cerebrospinal fluid escaped from the aperture. There was a rent in the dura. A "smooth recovery" was reported.

5. Briani [29] had a case in which, through the base of a circular opening "as large as a ten-cent piece" left by operative procedures, the dura mater, of perfectly normal appearance, projected. He reported "complete recovery in twenty days."

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78. Petterino Patriarca, A., and Rosello, G.: Osteoma primitivo del seno frontale concomitante a mucocele fistolizzato, Riv. oto-neuro-oftal. **11**:153-161, 1934.

79. Schneirer, J.: Ein Fall von Stirnhöhle-Mukozele mittels operativen Durchbruches gegen die Nase geheilt, Wien. med. Wchnschr. **76**:233, 1926.

6. Gaus's [17] case, published in 1935, that of a soldier whose right eye had been destroyed by a gunshot wound and his left enucleated because of shrapnel injury, showed interesting complications of the dura. After enucleation of the left eye, recurrent abscesses and intermittent protrusion of the area of the frontal sinus occurred. Over a period of fifteen years three further operations were necessary, radical operative procedures being contraindicated because of involvement of the dura. In the third operation a cystic tumor was revealed, the posterior wall of which was formed by very thin dura. More fragments of shrapnel were removed. As the surgeon proceeded, an oval aperture appeared in the posterior wall (dura), and cerebral fluid began to ooze into the field of operation. Further surgical intervention was deemed dangerous, and attention was directed toward healing of the dura. This was accomplished in eight days. The report was "recovery very good."

7. In Valerio's [76] case a tumor as large as a hen's egg was found. Exploration after extirpation revealed a cavity occupying the orbit in its superior portion as far as the apex. The osseous formation of the roof had disappeared, so that with an instrument it was possible to discern the transmission of the pulsation issuing from the cerebral mass. Throughout the entire extent of the tumor the dura was intimately adherent to the wall of the mucocele.

8. In Bonnet's [41] case of giant mucocele, curettage of the frontal sinus disclosed a dehiscence 2.5 cm. wide at the top of the cavity in the osseous wall. At this point the dura mater was bare. Recovery was good.

9. Kelling's [80] patient had a tumor as large as a hen's egg, which had perforated the orbital roof and broken into the orbit, causing almost complete erosion of the anterior, lower and posterior walls of the frontal sinus. The tumor had also broken into the ethmoid cavity. The dura mater was exposed in an area about as large as a "two-mark piece." "Permanent recovery" was reported.

10. Sprenger [81] described in great detail a case in which operative exploration revealed an aperture in the upper wall of the frontal sinus, through which the dura mater, which appeared normal, was exposed in an area the size of "a fifty-pfennig piece." He reported "complete recovery."

11. In case 3 of Paunz,[52] both the dura and the orbital contents lay freely exposed.

12. Jacques and Gamaleia [82] reported a case in which the cerebral walls (roof) and sinus floor presented wide lacunas, through which could be seen the cerebral palpitations and the orbital contents.

13. In Zentner's [67] case operation revealed a mucocele extending to the tip of the orbit, with the dura completely exposed throughout practically the entire extent of the orbital roof, so that pulsation of the brain could be clearly seen. The condition was so precarious that a radical operation was contraindicated.

14. In a case in which operation was done by Gibb,[83] the septum, the inner plate, the anterior wall of the frontal sinus and the orbital plate were eroded and

80. Kelling, G.: Analyse des Inhaltes einer Schleimcyste der Stirnhöhle, Wien. med. Wchnschr. **52**:1528-1530, 1902.

81. Sprenger: Ein Fall von Schleimhautcyste der Stirnhöhle, Arch. f. Laryng. u. Rhin. **19**:136-144, 1906.

82. Jacques, P., and Gamaleia, N.: Sur deux cas de mucocèle frontale, Ann. d. mal. de l'oreille, du larynx **41**:761-765, 1922.

83. Gibb, J. A.: Case of Pyocele of Frontal Sinus, Brit. M. J. **2**:900, 1921; Lancet **2**:1272, 1921.

the brain exposed. One must conclude from reading the report of that case that either a primary or a secondary infection was also present.

15. Wong [65] reported a case in which the lateral nasal wall was pushed inward and the floor and the medial wall of the orbit were absent. The lesser and the greater wings of the sphenoid were destroyed, and the optic nerve presented a stretched appearance. The dura at the floor of the orbit was exposed. The patient was reported cured.

16. In Bruzzone's [84] case 2 the patient showed, after operation, an exposed, reddened dura.

17-19. Dowman,[77] Garretson [35] and Fox [34] all noted absence of the posterior and the inferior bony walls of the frontal sinus, with exposure of the dura. In Fox's case the anterior bony wall also was lacking.

TREATMENT

It may be helpful to give references to a few outstanding contributions to the literature on the therapy of mucocele. Boenninghaus [1] in all his articles on the subject dealt with the therapeutic aspects. Benjamins [58] compared indications for internal and external operative procedures and reported an illustrative case of bilateral mucocele. Gignoux [85] contributed an interesting study of the evolution and treatment of mucocele, with outlines of 6 cases. Proby [86] described a case in which drainage of the frontal sinus by the nasal route was effected with the region under local anesthesia. Reverchon and Worms,[63] making a simple preliminary examination, accidentally perforated a very thin, fragile meatal septum with a stylet, which was sufficient to cure an external fistula and a mucocele. Rossier [87] described the treatment of an ethmofrontal mucocele, which for esthetic reasons was carried out by the endonasal route although an external incision seemed indicated. Gaus's [17] case of the soldier wounded by shrapnel is of special interest, since the report gives the technic of three operations, including the plastic procedure and the final operation for the application of Seiffert's permanent drainage.

From a historical point of view and as illustrations of some of the mistaken, painful and, to modern physicians, astonishing methods of treating mucocele employed in former times, the cases cited by Berthon [2] are of particular interest. One poor woman was treated by "the appli-

84. Bruzzone, C.: Sul mucocele dei seni paranasali, Arch. ital. di otol. **36**:379-394, 1925.

85. Gignoux, A.: Considérations sur l'évolution et le traitement des mucocèles du sinus frontal, Arch. internat. de laryng. **33**:513-526, 1927.

86. Proby: Mucocèle du sinus frontal droit, Lyon méd. **132**:498-501, 1924.

87. Rossier: Traitement endo-nasal d'une mucocèle ethmoïdo-frontale, Ann. d. mal. de l'oreille, du larynx **46**:49-53, 1927.

cation of two moxas (a role of combustible material or a redhot iron used on the skin to produce counterirritation) upon the crown of the head to cure a severe headache." As one would expect, "the wounds from these burns suppurated extensively," and finally, after thirty days of this agonizing treatment, relief came through death. It was found at autopsy, the report goes on to say, that the skull itself had been almost burned through.

In another woman, "discharge of pus was favored by emollient vapors directed into the nose through a funnel." Another patient, a man, was treated by "bleeding up to syncope, followed by the application of six leeches." Other forms of therapy equally bizarre could be cited if time and space permitted.[88]

REPORT OF CASE

A man of 36, presented himself on Sept. 22, 1932. He gave a history of childhood diseases. In August and September of 1929 he had had stubborn colds. Roentgenograms of his sinuses had been taken after each attack. In 1930 he had suffered an attack of influenza. During the first few days of September 1932 he had had acute bronchitis, which persisted for only a few days. No history of allergy was given.

Three days before the patient was first examined he had had a constant mild nonthrobbing headache over his left eye. It was never severe but persisted for two days. Some eighteen hours before he came for treatment this headache had suddenly ceased. He had then become aware of blurring of vision in his left eye.

Ocular Examination and Findings.—Vision at the time of examination was 20/13 in the right eye and 20/25 in the left eye. Examination showed the right eye to be essentially normal. The left eye presented no perceptible disease.

Dilatation of the pupils for a more complete examination was advised, but the patient postponed that for four days. His vision remained the same as on the first day until the fourth day, when that of the right eye was unchanged, while that of the left eye was less than 20/200. After dilatation of the pupils the right eye was found to be essentially normal and remained so throughout the patient's illness. It was impossible to obtain even a hazy view of the fundus of the left eye, because the vitreous was filled with small, rapidly moving vitreous opacities.

Physical Examination and Findings.—A complete, detailed physical examination, embracing roentgenograms, blood tests and a dental examination, was advised. The results of this examination were essentially negative. A Wassermann test was negative. A complete dental examination showed no abnormalities.

Sinus Examination and Findings.—A roentgen examination of the patient's sinuses on Aug. 19, 1929, had shown the following: The right frontal sinus was large and slightly cloudy. The left frontal sinus was very large and slightly cloudy. Just above the left orbit was an area of decreased density, which was attributed to possible destruction of bone. In the trabecula between the right and the left frontal sinus was a fair-sized osteoma. The left anterior ethmoid cells

88. The author on request will supply other references bearing on mucocele of the frontal sinus.

were slightly cloudy and the right anterior ethmoid cells very cloudy. Both right and left maxillary sinuses were moderately cloudy. The sphenoid area was cloudy, and the sella turcica was normal.

On Jan. 1, 1930, other roentgen examinations revealed the same picture. Another, on Sept. 26, 1932, showed the pathologic changes stated, as well as increased cloudiness of the left frontal sinus.

An immediate Lynch radical frontal sinus operation, ethmoidectomy and sphenoidectomy were advised.

Treatment.—Surgical Technic: With the patient under general anesthesia, the left frontal sinus was opened. The ethmoid labyrinth was disturbed as little as possible, as it was preferable to remove the infection of the frontal sinus before attacking the ethmoid or the sphenoid sinuses. After removal of the floor, the lining of the frontal sinus appeared normal in every detail, with the exception of an area adjacent to the nasofrontal duct. In this area, which was about the size of a nickel, the mucous membrane was considerably thickened. It was not edematous, nor did it show signs of infection but appeared to be merely thickened, otherwise normal mucous membrane.

The mucous membrane lining of the frontal sinus was stripped off and removed in the usual way. No undue effort was required. With the exception of the inner plate, the uncovered bone was normal in all details. No evidence of an osteoma was found. The inner plate was markedly eroded, and two large sequestrated pieces of bone were removed. This removal left little or none of the inner wall intact.

A portion of the dura approximately 1.5 cm. in diameter was necrotic. This was removed, exposing the brain substance. The cerebrum thus exposed showed no signs of infection, either acute or chronic. The vessels were not congested, no edema was noted and not even the slightest degree of herniation of the brain occurred. No cerebral spinal fluid was lost. Therefore, as would be expected, the dura was firmly attached to the brain substance around the exposed area. The remaining dura was normal in contour and appearance, with no visible signs of infection. Preoperative findings had indicated infection of the left anterior group of ethmoid cells, and surgical intervention proved this condition to be present.

If the ethmoid cells were disturbed further, what was the possibility that the infection would be scattered to such an extent that the dura or the brain substance would become infected? If, on the other hand, the infected ethmoid cells were not removed, would that be a factor minimizing in any way the possibility of success? The latter course, in spite of its dangers, was chosen. Had the ethmoid cells been exenterated, a large patent opening would have connected the frontal sinus with the left naris. To this my associates and I were opposed, despite the fact that some authorities state that the upper nasal passages are sterile.

But for the possibility of pus pockets developing, the external bony wall of the frontal sinus might have been removed, forcing the soft parts against the defect in the inner plate. The result of contact of infected material with the dura or the brain substance was also feared.

When the wound was closed, a large drain was inserted from the outside into the sinus at the inner angle. Skin clips were used to draw the margins of the skin together. The patient was put to bed apparently in excellent condition, his pulse rate being 76 and the respiratory rate 20.

Pathologic Findings.—Macroscopically, the tissue removed from the inner plate had the appearance of sequestrated bone. However, the pathologist stated that the specimens removed were really thickened mucous membrane.

Convalescence.—The patient had been admitted to the hospital on the night of September 27; thirty-three days later he was discharged, his recovery having been uneventful. At that time the drain was still in place and it was believed that the vision was normal in each eye.

Secondary Surgical Procedure.—When the drainage had almost subsided, the patient was readmitted to the hospital, fifty-one days after his operation. His white blood count and differential smear were normal. A urinalysis revealed no abnormality. The result of a general physical examination was negative.

On November 17 a secondary closure was done with the patient under general anesthesia. My associates and I had previously determined to examine the healing process of the frontal sinus, particularly in the region of the bony and dural defect, before closing the wound. Therefore, the drain was removed, and through the opening a nasopharyngoscope was passed. No evidence of infection was found. The walls were covered with healthy granulation tissue. The nasofrontal duct was enlarged and packed with gauze. The wound was then closed with skin clips.

Recovery.—The patient was discharged from the hospital on the fourth day after operation. Examination of the left eye showed that some swelling of the upper lid was still present; otherwise the eye was normal externally. The lens was normal. The dustlike vitreous opacities had entirely disappeared, although a few stringlike opacities were still present. However, in strong contrast to our inability just before the first operation to obtain even the slightest light reflex, we now had an excellent view of the fundus. No disease of the fundus was noted.

The patient had monocular vision in the mornings, when he was rested. Later in the day diplopia was very noticeable, making it impossible for him to read without covering one eye. With prismatic correction of the hyperphoria, he was quite comfortable.

At this time the patient, during a casual conversation, recalled that seven months prior to his first operation he had been awakened in the night by a discharge from his left nostril so profuse that it had saturated several handkerchiefs. This, together with the other findings, proved conclusively the diagnosis of mucocele. It also explained why the frontal sinus had appeared practically normal, with the exception of thickened mucous membrane, when it was opened and why no fluid was present.

At the end of two and a half years the muscular imbalance had entirely disappeared. The fundi were normal. After eight years the patient's general health is excellent, and he has had no further infections of his sinuses. No muscular imbalance or vitreous opacities are now present, and his vision is normal in each eye.

COMMENT

A mucocele of the frontal sinus may erode the inner plate, destroy the dura, pia and arachnoid and for a time produce no symptoms. Recovery after a correct surgical procedure is the rule.

A dural graft might have been inserted at the time of the operation had I been prepared for the condition which was found.

The orthodox closure in a case such as that reported here is the removal of the anterior plate of the frontal sinus, collapsing the soft parts against the inner wall. Despite this fact, there was less danger of an intracranial infection with the procedure employed in this case

In a case such as this the ethmoid labyrinth should not be disturbed more than is necessary.

The nasopharyngoscope was of definite value in examining the interior of the sinus before the secondary closure.

The Lynch radical frontal operation practically never causes any cosmetic deformity. In this case, despite the surgical modification employed, there was no exception.

A roentgen examination made eight years after the first operation revealed no abnormalities other than a slight haziness of the frontal and the ethmoid sinuses.

Case Reports

BACILLUS COLI BACTEREMIA WITH MASTOIDITIS AND SINUS THROMBOSIS

Report of a Case and Review of the American Literature

WILLIAM FELDMAN, M.D., NEW YORK

Otitic infections due to Bacillus coli or the related enteric organisms are rarely reported. Judging from the scanty literature, the mortality rate is high. It seems justifiable, therefore, to present another case of otitic infection due to B. coli, complicated by mastoiditis and sinus thrombosis, in which bacteremia supervened and recovery occurred.

REPORT OF CASE

E. S., a white man 30 years of age, came to my office on Sept. 28, 1932, complaining of pain in the right ear, slight aural discharge, mild headache on the right and general weakness.

History.—The onset of his complaints dated back four months, when he noticed a purulent discharge from the right ear. After treatment for six weeks, the ear became dry. Five days before seeing me he had chills, fever, nausea, earache on the right and a slight headache. These symptoms were severe enough to force him to go to bed. Since there was no remission, he came to see me. His temperature at this time was 101.6 F., and he was advised to be hospitalized. The same evening he was admitted to the New York City Hospital Otolaryngological Division, service of Dr. A. N. Schiller.

Physical Examination.—On admission he appeared acutely ill. He was cooperative, although somewhat sluggish mentally. Pertinent findings were confined to the right ear. The external canal was narrowed. There was a slight purulent discharge of rather offensive odor, tinged with blood. Pressure pulsations could be observed through a small perforation at the lower anterior segment of the thickened tympanic membrane. On deep pressure there was slight tenderness in the upper posterior part of the mastoid. Hearing on this side was impaired. The remainder of the physical examination gave negative results except the observation of a deviation of the septum, subacute inflammation of the tonsils and mild pharyngitis. The temperature was 102.6 F., the pulse rate 100 and the respirations 20. The urine showed albumin (2 +) and a few coarsely granular casts. The white blood count was 12,000, with 60 per cent mature polymorphonuclears and 32 per cent staff cells. The hemoglobin content and red cell count were normal.

On the next day the temperature ranged from 101 to 102 F. On the second day after admission the patient had several chills, lasting from ten to fifteen minutes each. By noon his temperature had risen to 107 F. He was vomiting, complained of headache on the right and of earache and appeared septic. The neck was slightly rigid, there was a suggestion of a Kernig sign and petechiae appeared on the abdomen. A spinal tap revealed a clear fluid under increased pressure; the spinal fluid showed 140 cells per cubic millimeter, with increased sugar and a

Read before the Bronx Otolaryngological Society, Jan. 28, 1941.

From the Otolaryngological Service, New York City Hospital, Welfare Island.

trace of albumin. A Tobey-Ayer test revealed no rise in spinal fluid pressure when the right jugular vein was compressed and a normal increase with pressure on the left jugular vein. A culture of the spinal fluid was negative. Roentgen examination of the mastoids showed a diffuse pathologic process on both sides, somewhat more dense on the right than on the left, which was interpreted as osteosclerosis. On the right side only a small portion lying in front and behind the lateral sinus showed pneumatization. The sinuses were well outlined on both sides. Examination of the ocular fundi revealed early bilateral papillitis.

Operation.—On the same afternoon a mastoidectomy was performed on the right. The mastoid was dense and sclerotic, with no cellular structure except a few cells bordering the sinus plate. As the sinus plate was reached, there exuded from a small opening a foul-smelling pus, which bubbled as the opening was enlarged. This opening was found to extend to the sinus wall. The sinus plate was uncovered from the knee down to the jugular bulb. The sinus wall was found to be collapsed and necrotic for about 2 cm. There was no bleeding from above or below. The necrotic area of the sinus wall was excised. Further exposure of the lateral sinus toward the torcular Herophili, with removal of an obturating thrombus, produced bleeding from above, but no bleeding was obtained from the jugular end of the sinus. The right jugular vein was ligated above the facial vein.

Postoperative Course.—The patient made an uneventul recovery. The temperature became normal in eight days. He was discharged from the hospital on October 29, thirty-one days after his admission.

Bacteriology.—Prior to operation blood was taken for culture. This revealed the presence of a nonmotile gram-negative bacillus of the colon group. In aerobic culture the organism was a slow lactose fermenter and a rapid saccharose fermenter; in anaerobic culture it proved proteolytic. It was concluded to be an atypical colon bacillus, falling into a classification between the typical colon organisms and the proteus group.

At operation, cultures were made of the pus found in the mastoid and of material taken from the sinus. The same type of organism which had been recovered from the blood stream was recovered from these two sites.

A blood culture made three days after operation was negative.

It seems to me that this was a case of ambulatory disease and that a localized abscess was found in the lateral sinus at operation. Nature was kind to the patient in sealing off the proximal and distal ends of the sinus with a protective thrombus, until a reinfection had taken place, followed by an exacerbation of symptoms. The finding of the unusual organism is extremely interesting.

Infections of the middle ear, mastoid and sinuses with enteric organisms like B. coli and Bacillus proteus, with or without septicemia or bacteremia, are infrequently reported in the American literature.

Apparently, the first case reported was that of Dougherty,[1] in 1914. He stated that he could find no previous reference in the American literature. His patient was a youth aged 20 with a history of purulent otitis on the right of two months' duration. A short time previously he had been operated on for an ischiorectal abscess. The patient was

1. Dougherty, D. S.: Colon Bacillus Infection in Middle Ear Disease, New York M. J. **100**:1163-1165, 1914.

beginning to show signs of intracranial involvement, and surgical intervention was advised. A radical mastoidectomy was performed on the right side. Culture of material from the mastoid showed large numbers of colon bacilli, a few streptococci and a gram-positive bacillus. Five days later symptoms of sinus thrombosis developed. The mastoid wound was revised and the sinus exposed from the torcular Herophili to the jugular bulb. Below the bulb the sinus was found thrombosed for about 1 inch (2.5 cm.). Culture of material taken from the wound showed colon bacilli and streptococci. The patient died. Autopsy was not performed.

Dougherty reported another case of acute mastoiditis with an intracranial complication. Mastoidectomy was performed. The pus of the mastoid yielded colon bacilli in pure culture. The patient died. Autopsy revealed an abscess in the left temporal lobe. I could find in the literature only one other reference to an intracranial complication due to colon bacillus, that in the report of Libman.[2] Among 141 cases of aural discharge or complications of otitis media, he found the colon bacillus in 1 case of abscess of the brain. Dougherty also reported 4 cases of mastoiditis following otitis media. In each case a mastoidectomy was performed and a pure culture of bacillus colon was obtained from the pus of the mastoid. It is interesting to note that 3 of his patients had furunculosis of the external canal before the middle ear became involved. The other had an ischiorectal abscess and later otitis media followed by mastoiditis. In his cases the possibility of the infection's having been spread to the middle ear and mastoid by the scratching of an abraded surface with a fecally contaminated finger is considered.

Warren and Herrick [3] reported the case of a patient 28 years of age, in which the diagnosis was mastoiditis and bacteremia. A blood culture revealed the colon bacillus, 1 per centimeter. A mastoidectomy was performed, and the patient recovered.

Mygind [4] reported that in 4 of his series of 70 cases of otogenic sinophlebitis the colon bacillus was found in pure culture. He did not state, however, where he procured the material for culture—whether from the ear, mastoid or blood.

Smith [5] reported the case of a woman aged 36 with acute fulminating mastoiditis. The patient gave a history of hematuria and dysuria one year before and of recent nocturia, necessitating urination two or three times during the night. The illness reported was complicated by bronchopneumonia. A mastoidectomy was performed with the region under local anesthesia, and the lateral sinus was explored. In the sinus was found a purulent clot, which extended from the jugular bulb to the

2. Libman, E.: Value of Bacteriological Investigation in Otology with Special Reference to Blood Cultures, Arch. Otol. **37**:22, 1908.

3. Warren, M., and Herrick, W. W.: Analysis of One Hundred and Thirty-Four Cases of Bacteraemia, Am. J. M. Sc. **151**:556-577 (April) 1916.

4. Mygind, H.: Contribution to the Etiology and Pathogenesis of Otogenic Sinus Phlebitis, J. Laryng., Rhin. & Otol. **31**:497-515, 1916.

5. Smith, C. H.: Aural Infections from Colon Bacillus: Report of a Case, Arch. Otolaryng. **19**:249-251 (Feb.) 1934.

torcular Herophili. Free bleeding was obtained from the torcular end. On account of the local anesthesia, the jugular vein was not exposed until five days later, at which time it was found that the vein contained pus, and a resection of the internal jugular vein above the facial vein was performed. Culture of blood taken on the day of operation showed the presence of B. coli. Bacteriologic examination of the pus from the mastoid, the lateral sinus and the internal jugular vein also showed the presence of B. coli. Several later blood cultures were positive for the same organism. B. coli phage was given intravenously daily. After a stormy course the patient died.

Necropsy revealed an abscess in the pelvis of the kidney, meningeal inflammation and extensive bronchopneumonia. The pus from the kidney, when cultured, showed B. coli communis. This was a case in which aural infection with bacteremia was secondary to a renal infection, although clinical evidence gave no proof of the later infection and repeated urinalyses were negative.

Atkinson [6] reported the case of a boy 10 years of age who had an acute exacerbation of chronic otitis media. At operation the lateral sinus was found thrombosed, and a cerebellar abscess was drained. Bacteriologic examination showed Streptococcus pyogenes and B. coli communis.

Gerzog [7] reviewed 9 cases of otogenous bacteremia due to B. proteus, which he gathered from the literature since 1912, and added a case of his own. Seven of the patients gave a history of chronic otitis; all were operated on, and 4 had the lateral sinus obliterated and the internal jugular vein ligated. In all but 1 case the blood cultures were positive, and the patient in that case and his own patient recovered. Gerzog attributed the recovery of his patient to the use of a bacteriophage and suggested its use against similar infections. The mortality rate in this series was 89 per cent; 3 of the patients died of meningitis and 5 of overwhelming bacteremia.

Brownell [8] reported the case of a child 11 years of age with a history of intermittent bilateral otorrhea of two years' duration. Subsequently the patient had a chill, projectile vomiting and stiffness of the neck, and there was tenderness to pressure over the right internal jugular vein. Ophthalmologic and neurologic examination gave negative results, and a blood culture did not show any growth. A radical mastoidectomy was performed on the right side. The internal jugular vein was found thrombosed, and the lateral sinus was found to be filled with an old clot. Free bleeding was not obtained from either end. A culture of the pus inside the sinus demonstrated B. coli. Empyema and a pulmonary abscess developed. B. coli was cultured from the sputum. After a stormy course the patient recovered.

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6. Atkinson, E. M.: Treatment of Lateral Sinus Infections Without Operation, Arch. Otolaryng. **27**:169-177 (Feb.) 1938.

7. Gerzog, B. G.: Bacillus Proteus in Otogenic Infections Secondary to Mastoiditis, Thrombosis of the Lateral Sinus and Bacteremia, Arch. Otolaryng. **30**:275-279 (Aug.) 1939.

8. Brownell, D. H.: Unusual Cases of Thrombosis of the Sigmoid Sinus, Arch. Otolaryng. **31**:663-668 (April) 1940.

Goldman,[9] in his series of 104 cases of bacteremia with thrombosis of the lateral sinus, reported blood cultures positive for B. proteus in 2 cases.

SUMMARY AND CONCLUSIONS

A case of mastoiditis complicated by sinus thrombosis with bacteremia, in which the infecting organism was the colon bacillus, is presented. The patient recovered after surgical intervention.

A clinical analysis of colon bacillus bacteremia in otogenic infections reveals that it is relatively uncommon as compared with bacteremia due to other pathogens and has a high mortality rate.

The presence of thrombosis and necrosis of the lateral sinus suggests that the colon bacillus has an affinity for vascular walls. The cases reported in the literature seem to support this suggestion, as they also show a tendency to the same type of involvement of the sinus.

Dr. J. R. Lisa, pathologist at the New York City Hospital, Welfare Island, contributed suggestions and criticism to the preparation of this paper.

2021 Grand Concourse.

9. Goldman, J. L.: Blood Cultures in Cases of Otitic Sepsis, Arch. Otolaryng. **30**:516-524 (Oct.) 1939.

OTITIC MENINGITIS DUE TO BACILLUS PROTEUS

NELSON R. CRAGG, M.D., CINCINNATI

Bacillus proteus is an actively motile gram-negative organism which grows readily on ordinary mediums at room temperature. It is hemolytic and proteolytic. It is widely distributed in nature, being found in almost all putrefactive matter of animal origin, in soil contaminated by animal excretions, in stagnant pools and in sewage. It is commonly found in the mouth, on the skin and in the urine and feces of normal persons; however, this organism is capable of producing severe infections. Its presence has been reported frequently in such conditions as otitis media, cystitis, pyelitis and war wounds and rarely in meningitis.

In 1922 Kernan [1] reported 2 cases of B. proteus infection, in one of which the infection originated from a peritonsillar abscess and in the other, from chronic otitis media associated with an epidural abscess. A positive blood culture was obtained in the latter case. Both cases terminated fatally.

In 1924 Warren and Lamb [2] described a case of B. proteus septicemia originating from an ulceration of the soft palate. At autopsy, ulcerations of the intestinal mucosa were also found.

In 1927 Neal and Abramson [3] reported 7 cases of meningitis due to B. proteus, in each of which the spinal fluid yielded a pure culture of the organism. In only 2 of these cases could the meningitis be proved to be secondary to otitis media. One of the patients recovered.

In 1928 Taylor [4] described 4 cases in which otitis media was followed by meningitis due to B. proteus. Brain abscess developed in 2 of the patients. A fifth patient with chronic otitis media and mastoiditis recovered.

In 1929 King and Morgan [5] reported B. proteus septicemia in a tuberculous patient having multiple pulmonary abscesses. Positive cultures of B. proteus were obtained also from the frontal and maxillary sinuses and from the sputum.

In 1929 Buckley [6] described 2 cases of B. proteus septicemia, in one of which the infection followed suprapubic prostatectomy and in the other, dilatation of an anterior urethral stricture.

In 1929 Kretschmer and Mason [7] reported 2 cases of kidney stone in which blood culture yielded B. proteus. Both patients recovered.

From the Department of Otolaryngology of the Cincinnati General Hospital.

1. Kernan, J. D.: Laryngoscope **32**:304-310 (April) 1922.
2. Warren, S., and Lamb, M. E.: J. M. Research **44**:375-381 (March) 1924.
3. Neal, J., and Abramson, H.: Am. J. M. Sc. **174**:665-670 (Nov.) 1927.
4. Taylor, J. F.: J. Path. & Bact. **31**:897-915 (Oct.) 1928.
5. King, M. J., and Morgan, R. H.: Am. Rev. Tuberc. **19**:182-190 (Feb.) 1929.
6. Buckley, T.: Lancet **2**:530-531 (Sept. 7) 1929.
7. Kretschmer, H. L., and Mason, L. W.: Bacillus Proteus Septicemia: Report of Two Cases with Recovery, J. A. M. A. **92**:1734-1737 (May 25) 1929.

In 1930 Stein [8] reported 3 cases of B. proteus infection. One patient had simple recurrent mastoiditis, with recovery. The others had thrombosis of the internal jugular vein, resulting in death.

In 1930 Walters and Scott [9] described a case in which B. proteus septicemia followed the first stage of suprapubic prostatectomy, with recovery.

In 1932 Paryzek and Ecker [10] isolated B. proteus from a patient with postpartum septicemia, who recovered. This patient had chills and high fever for two months.

In 1935 Smith and Miller [11] reported a case in which chronic otitis media resulted in thrombosis of the lateral sinus and terminated fatally. A positive blood culture was obtained in this case.

In 1936 McGovern [12] described 2 cases of B. proteus infection. In both cases infection originated from chronic otitis media and resulted in thrombosis of the lateral sinus and death. Blood cultures were positive, but cultures of the spinal fluid were negative.

In 1936 Calhoun [13] reported a case in which meningitis due to B. proteus originated from chronic otitis media. A culture of the spinal fluid was positive. A radical mastoidectomy was done, and the patient recovered.

REPORT OF A CASE

A patient was admitted to the Cincinnati General Hospital on May 8, 1939, with a history of pain in her left ear for the past three days, associated with headache, and severe pain in the neck on attempting to move her head. The left ear had been draining intermittently for the past two years. The temperature on admission registered 104 F.

Physical examination revealed a well developed and well nourished white girl aged 15, acutely ill. There was a profuse discharge of foul-smelling pus from the left ear and moderate tenderness over the mastoid. Neurologic examination disclosed slight facial weakness on the left side, associated with mild ptosis of the left eyelid, rigidity of the neck, and the Kernig and the Brudzinski sign.

The laboratory findings on admission were as follows: The red blood cells numbered 3,840,000 and the white blood cells 18,000 per cubic millimeter. The differential blood count revealed 89 per cent polymorphonuclears, 10 per cent lymphocytes and 1 per cent monocytes. Urinalysis showed 15 to 25 white cells per high power field. The spinal fluid was cloudy. The Pandy reaction was 3 plus. Chemical analysis showed proteins 113 mg. per hundred cubic centimeters, sugar 30 mg. and chlorides 694 mg. A smear showed a gram-negative rod.

Operation.—The patient was operated on shortly after admission on May 8. A sclerotic mastoid was encountered, associated with a perisinal abscess and cholesteatoma. The pus was brownish and foul smelling, and the lateral sinus was covered with granulations. A modified radical mastoidectomy was done. The administration of sulfanilamide was started immediately after the operation. On

8. Stein, S.: Ann. Otol., Rhin. & Laryng. **39**:1155-1160 (Dec.) 1930.

9. Walters, W., and Scott, D. E.: Proc. Staff Meet., Mayo Clin. **5**:168 (June 18) 1930.

10. Paryzek, H. V., and Ecker, E. E.: Am. J. M. Sc. **184**:533-536 (Oct.) 1932.

11. Smith, A. B. P., and Miller, S.: Lancet **1**:862 (April 13) 1935.

12. McGovern, F. H.: Fatal Otogenic Infection with Bacillus Proteus: Report of Two Cases, Arch. Otolaryng. **24**:618-621 (Nov.) 1936.

13. Calhoun, J.: Canad. M. A. J. **34**:679-680 (June) 1936.

May 10 the laboratory reported that cultures of B. proteus had been obtained from the blood, the spinal fluid and material removed from the external ear and from the mastoid. As the patient continued to have a high fever associated with chills, the lateral sinus was inspected again and was found to be thrombosed. On May 11 the internal jugular vein was ligated and the thrombus was removed from the sigmoid sinus distally until free bleeding was obtained. The thrombus extended into the jugular vein to the middle thyroid vein. Because of the weakness of the seventh nerve and the paralysis of the sixth nerve, petrositis was suspected. On May 13 the petrous tip was explored but no signs of infection were encountered. The patient improved greatly after the ligation. The temperature became normal, the spinal fluid cleared, the blood cultures became negative and the operative wounds were healing nicely. Because of this improvement the administration of sulfanilamide was discontinued after seven days of therapy.

On May 21 a cough developed, which soon became productive of a brownish foul-smelling sputum. Culture of the sputum revealed Pneumococcus type XIX. The administration of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) was started, continued for five days and then stopped because the clinical course was progressively down hill in spite of almost daily transfusions. Culture of the sputum also showed B. proteus. Medical and surgical consultations were obtained, and a diagnosis of lung abscess and empyema was made. The patient was subsequently transferred to the surgical service. On May 28 thoracentesis was done and 850 cc. of dark amber fluid was obtained. A culture of this fluid was negative. On May 31 thoracentesis was repeated, and this time B. proteus mixed with Staphylococcus and Streptococcus was obtained from culture. On June 5 thoracotomy was done by means of a trocar. Resection of the ribs was delayed until June 12 on account of the patient's extremely serious condition. It was done in successive stages, according to the amount of surgical procedure the patient could stand, on June 12, June 28 and August 29.

From the onset of the lung abscess to the beginning of convalescence (four weeks) the patient's course was stormy, with frequent chills and fever. The administration of sulfanilamide was again started on June 3 and was continued through June 27. The patient remained in the hospital until November 4.

Several weeks postoperatively a cerebellar fungus developed at the site of the mastoid operation. It increased in size for several weeks but finally receded after the grafting of skin. When the patient was discharged from the hospital, on November 4, the fungus had disappeared. The left ear continued to discharge slightly during the entire stay in the hospital. The patient is being followed in the clinic. The ear is dry.

SUMMARY

A case of meningitis due to B. proteus and secondary to chronic otitis media and mastoiditis, associated with thrombosis of the lateral sinus and lung abscess, is reported. The organism was cultured from the blood, the spinal fluid and material removed from the ear, the mastoid and a pulmonary abscess. Treatment consisted of mastoidectomy, ligation of the internal jugular vein, resection of ribs, administration of sulfanilamide and sulfapyridine and transfusions.

COMMENT

B. proteus is a frequent inhabitant of the middle ear and of the mastoid when there is a chronically draining ear, but it rarely becomes pathogenic. In most instances the organism is found in combination

with Streptococcus and Staphylococcus. In the literature up to the present, only 12 cases of meningitis due to B. proteus have been recorded, and in only 2 did the patients recover. In 8 of the 12 cases meningitis was secondary to otitis media, in 2 it originated from peritonsillar abscesses complicated with thrombosis of the internal jugular vein and in 2 it was of undetermined origin. Thirteen cases of septicemia due to B. proteus have also been recorded in the literature, in 5 of which the patients died. From a single case one cannot determine the value of sulfanilamide or sulfapyridine in combating B. proteus infection. However, it was the consensus of those associated with this case that the patient would not have recovered without chemotherapy.

2600 Union Central Building.

NASAL FOREIGN BODIES

JOSEPH C. DONNELLY, M.D., PHILADELPHIA

The nature and the removal of the average nasal foreign body hardly warrant a report in the literature. In the case recorded here, however, the roentgen diagnosis of the presence of a foreign body and the subsequent removal of the body as an unrecognizable mass appeared sufficiently interesting and instructive to prompt publication.

SYMPTOMS AND DIAGNOSIS

As a rule, it is easy to detect the presence of a foreign body in the nose, but occasionally the symptoms are so slight that they escape notice. A 6 year old child, as recently reported by Kopp and Levy,[1] was treated intermittently for three years because of repeated head colds. Adenoidectomy and tonsillectomy failed to help. Later a frontal headache and a foul nasal discharge developed. A roentgenogram taken to determine the condition of the paranasal sinuses showed a metal button wedged between the septum and the posterior tip of the inferior turbinate.

The question of a foreign body in the nose might not be considered by the attending physician if the main symptoms were referred to the lower respiratory or digestive tract, but such problems were encountered by Poe,[2] with the nasal symptoms playing only a minor role. A child 3½ years of age suffered from repeated attacks of bronchial asthma and occasionally had some bleeding from the nose. The wheezing stopped shortly after the removal of a piece of putty from the nasal fossa. Another child, of 2 years, had been troubled with anorexia, vomiting and diarrhea for three months, while a nasal obstruction appeared a trifling matter. However, the removal of a fuzzy weed from the nose was followed by a cure of the gastroenteritis.

Foreign bodies in the nose, like foreign bodies in the lung, may occasionally be discovered accidentally by either the clinician or the roentgenologist. Camerer,[3] in the routine clinical examination of a number of patients, came across a foreign body in a 22 year old seaman who had no symptoms or totally disregarded them despite the fact that his nose had been host to a calcified cork for seventeen years. Inquiry revealed that he dimly recalled having his nose examined by

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From the Department of Otolaryngology of the University of Pennsylvania.

Read before the Section on Otolaryngology of the College of Physicians of Philadelphia, Oct. 16, 1940.

1. Kopp, M. M., and Levy, H.: Foreign Body in the Nose (Report of Case), Laryngoscope **48**:819-821 (Nov.) 1938.

2. Poe, D. L.: Foreign Bodies in Children's Noses and Ears, Arch. Pediat. **54**:434-440 (July) 1937.

3. Camerer, C. B.: Foreign Bodies in Nares, U. S. Nav. M. Bull. **10**:168-169 (Aug.) 1923.

a physician at the age of 5 because there was a question at that time of something having been introduced into his nose by him or a playmate.

Before resorting to a roentgen examination of an adult suspected of harboring a nasal foreign body, it might be wise to ask about previous surgical treatment to the nose or exodontia. In the experience of Jones,[4] a patient complained of a sense of fulness in the right side of the face and a postnasal dripping of fetid pus. Several physicians had been consulted, and dentists advised the extraction of some upper molar teeth. The roentgen study of this patient disclosed a piece of a nasal curet in the right antrum, which had been operated on two years previously. A unilateral fetid discharge is sometimes the aftermath of exodontia, as was shown by Hargett[5] in describing the condition of a nurse in whom facial pain and nasal discharge developed after the extraction of an upper molar tooth. After the fifth irrigation of the affected antrum, the patient became conscious of a mass in the nose, which proved to be a piece of gauze used in packing the tooth socket.

The typical findings of nasal obstruction and unilateral fetid discharge were present in the interesting case reported by Wolfe[6] a few years ago. The patient's nose harbored the blade of a penknife for ten years, the symptoms dating from an accident. Despite the long sojourn of the foreign body there was but little damage to the surrounding nasal tissue.

REPORT OF A CASE

A girl, T. D., 7 years old, was referred to the University Hospital for adenoidectomy and tonsillectomy, with the hope that her breathing, appetite and general health would be improved. For the previous two years she had had difficulty in breathing through the right nostril, and she was frequently troubled with a frontal headache. At times she complained of tenderness over the right side of her nose when her face was being washed. No discharge or odor had been observed.

Examination showed that the left nasal fossa was free of secretion, but on the right side a mass could be seen coming out of the middle meatus and impinging on the septum. Covered with a thick and tenacious grayish exudate, it first resembled a fibrous polyp. The whole picture was not unlike that of suppurative and hyperplastic sinusitis. The character of the exudate, however, was not typical of pus, and on palpation with a probe the mass felt very firm. Suspicion was aroused then as to the presence of a foreign body, and the patient was referred for a roentgen examination, which revealed in the right nasal fossa a metal screw (fig. 1), approximately 5/8 inch (1.6 cm.) long, with the head pointing anteriorly. Since it was anticipated that the screw might be impacted and that extraction would be attended with difficulty, divinyl ether (vinethene) was administered to the child as an anesthetic in the accident ward, where an attempt was made at

4. Jones, W. I.: Foreign Objects in Antrum of Highmore, Am. J. Orthodontics **25**:380-382 (April) 1939.

5. Hargett, E. R.: Expulsion of Foreign Body from Antrum with Aid of Irrigation, Arch. Otolaryng. **31**:332 (Feb.) 1940.

6. Wolfe, J. J.: Unusual Foreign Body in the Nose, Arch. Otolaryng. **26**: 736-737 (Dec.) 1937.

removal. A firm grasp and considerable traction on the presenting head of the screw removed only the immediate part within the mouth of the forceps. In size, shape and color the mass resembled a black shoe button. The second black and

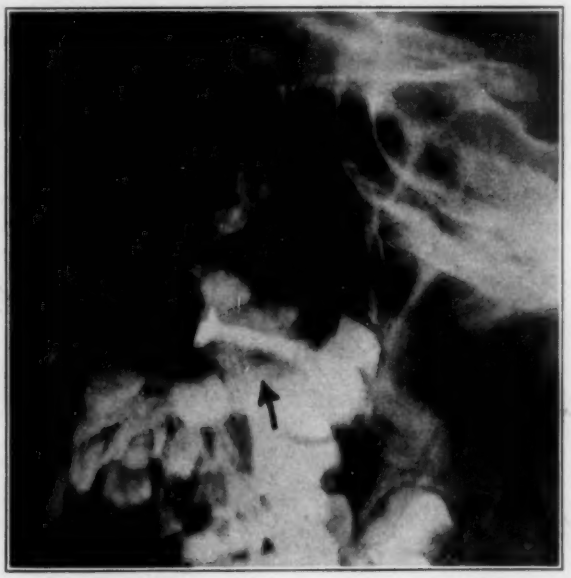


Fig. 1.—Lateral roentgenogram, showing the screw in the nasal fossa.

Fig. 2.—Photograph of unrecognizable particles of the screw.

brittle piece which was dislodged could be crumbled easily between the finger tips. Considerable bleeding followed; so the child was admitted to the hospital In the operating room with the patient under ether anesthesia, the remaining corroded particles (fig. 2) were removed in piecemeal fashion by the use of suction and a small ring curet. The exuberant granulations which had practically encapsulated

the foreign body were curetted from the nasal mucosa. A second roentgenogram (fig. 3) proved that the screw had been removed entirely. The anteroposterior view of the paranasal sinuses showed a definite clouding of the right antrum and right ethmoid cells. This apparently was the aftermath of impaired ventilation rather than a true suppuration, because the nasal inflammation rapidly subsided and subsequent clinical examination failed to show any abnormal secretion indicative of sinal disease. The child was permitted to return home on the following day. Her appetite immediately improved as her headache and the tenderness over the right side of her nose disappeared.

COMMENT

It was evident from the corroded and crumbled mass removed at operation that the metal screw would not have been identified had we

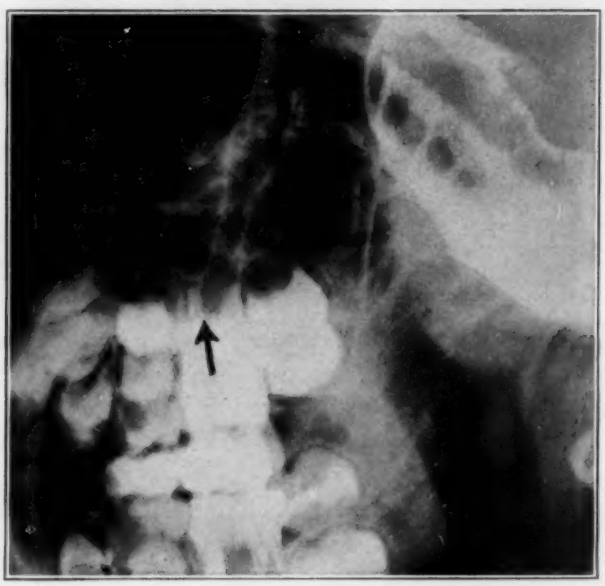


Fig. 3.—Lateral roentgenogram made after the screw was removed.

not had a previous roentgenogram. The finding of such a charred black mass exactly duplicates the experience of the Jacksons,[7] who occasionally have described foreign bodies, like nails and screws, removed from the lung as a mere mass of corroded material resembling coke. They have noted that certain metals after a long sojourn in the lung lose their tensile strength and are easily crumbled when pulled on by a forceps. They have observed further that galvanized coated objects lose their coating in one and a half years, the corrosion occurring rapidly at first and more slowly as the crust of corroded material accumulates. The screw described here was present in the child's nose

7. Jackson, C., and Jackson, C. L.: Diseases of the Air and Food Passages of Foreign-Body Origin, Philadelphia, W. B. Saunders Company, 1936.

for two years, and from this one may assume that the corrosive action of the nasal secretions on metallic substances simulates the chemical changes which occur in the lung.

SUMMARY

The presence of a metal screw in the nose of a child for two years without any perceptible discharge or odor has been described.

Points in the diagnosis of the presence of nasal foreign bodies have been reviewed, attention being directed to the main symptoms, which occasionally are referred to the lower respiratory or digestive tract.

A roentgenogram may be the only means of identifying a foreign body which has remained in the nose over a long period.

The chemical decomposition of certain metals in the nasal cavity simulates the changes in the lung observed by endoscopists.

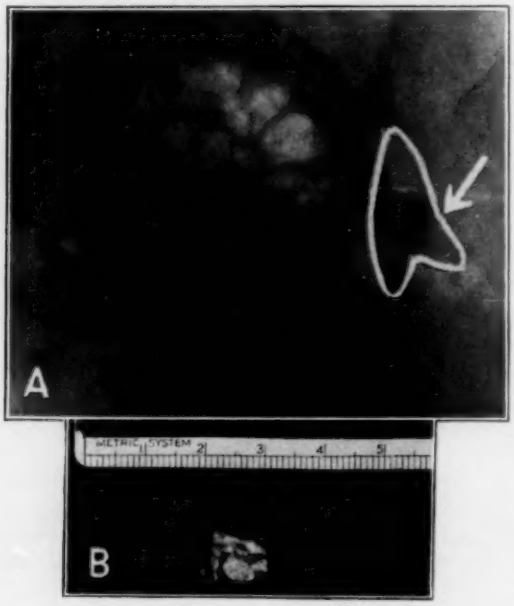
2008 Walnut Street.

BONY DERMOID CYST OF THE EXTERNAL AUDITORY CANAL RESEMBLING A DECIDUOUS TOOTH

SAMUEL KAPLAN, M.D., AND JOHN J. DONLON, M.D., BROOKLYN

The following case is reported because of its unusual interest and because a similar one does not seem to have been recorded in the literature.

W. S., a white youth aged 20, came with the following complaint: He tried to enlist in the United States Navy and was rejected because of a bony tumor in the left auditory canal. Prior to this the patient had been completely unaware



A, roentgenogram of the left external auditory canal. *B*, photograph of the specimen.

of the tumor. Examination revealed a hard mass covered by uninterrupted skin, protruding from the anterior superior wall of the left external auditory canal at about the junction of the cartilaginous and the bony portions. The mass reduced the lumen of the canal by two thirds. However, inspection with a small speculum showed a normal drum membrane behind it. Hearing tests gave negative results. Roentgenograms showed an irregularly triangular density, of which one corner protruded into the auditory canal and the remainder lay in or in front of the lateral mastoid and squamous cortex (fig., *A*).

The patient was admitted to the hospital on Oct. 21, 1940 and operated on the same day. With the region under local anesthesia induced with procaine hydrochloride, a small incision was made in the skin over the bony mass; the overlying

From the Otorhinolaryngology Service, Long Island College Hospital.

skin was then removed by means of a small curet. Simple pressure on the curet shelled out a small bony mass from the anterosuperior wall of the canal. The postoperative course was uneventful, with the cavity healing completely within two weeks.

Comment.—The bony mass removed had the structure of a deciduous tooth and was identified as such by Joseph Kaplan, D.D.S. However, the specimen was sent to Dr. John M. Pearce for microscopic examination, and the following report was submitted by him:

"Nature of specimen: deciduous tooth. Gross description: The specimen (fig., *B*) is an irregular bony-hard mass measuring 1 by 1.8 by 0.5 cm. Adherent to it is a small amount of dense fibrous tissue.

"Microscopic description: The specimen consists of a mass of dense bone made up of broad anastomosing trabeculae with few and small spaces between them. In these spaces, however, a variety of structures are scattered throughout a loose fibrous stroma. Most prominent are cystic spaces lined by stratified squamous epithelium containing desquamated sebaceous plates. An occasional hair follicle can also be found. Another type of cystic space is filled with multinucleated giant cells. Along the edges of the bone and sometimes within the spaces formed by it is a layer of squamous epithelium. On this and often far from the surface are structures closely resembling sweat glands and sebaceous glands.

"Diagnosis: dermoid cyst."

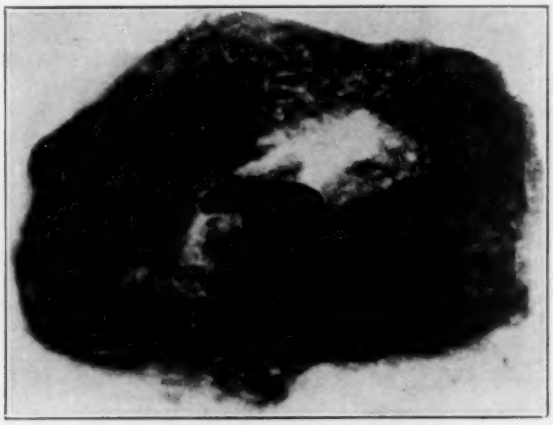
Clinical Notes; New Instruments and Technics

A NEW METHOD FOR INHIBITING OSTEOGENESIS IN ARTIFICIAL BONE FISTULAS

Preliminary Report

PAUL GUGGENHEIM, M.D., AND LOUIS GUGGENHEIM, M.D., LOS ANGELES

In a previous paper [1] were reported the results of experiments with three methods of producing fistulas in bone, viz. (1) with a sharp chisel, (2) a burnishing burr and (3) a burnishing burr followed by electrocoagulation. After two months and two days there was evidence of



Fistula made with a sharp chisel and treated with copper sulfate; on one wall there is a filament of copper wire attached to a projecting portion of bone.

bone formation where each of the three methods had been used, but where the sharp chisel had been employed, the osteogenesis was least marked

The present research was undertaken with the thought that through chemical means it might be possible to inhibit osteogenesis.

It is known that copper, even in minute amounts, inactivates vitamin C by catalytic oxidation. Vitamin C is necessary to the gelling of collagen in the formation of bone matrix; therefore in the absence of vitamin C bone cannot form.

As in our previous work, the fistulas were made in the craniums of white rabbits. Three experiments were undertaken; in all the fistulas were made with a sharp chisel.

From the Department of Otolaryngology of the University of Southern California.

1. Guggenheim, L., and Guggenheim, P.: Artificial Fistula in Cases of Otosclerosis, Arch. Otolaryng. **32**:1-15 (July) 1940.

In the first experiment the osseous fistula was treated with saturated solution of copper sulfate, and a fine filament of copper wire was tied around a projecting portion of bone.

In the second experiment a small fistula was treated with saturated solution of copper sulfate; a larger control fistula was left untreated.

In the third experiment a filament of copper wire was tied to an osseous projection in a large fistula, while a small fistula was left untreated.

The first experiment was interrupted at two months and eleven days. The specimen was removed with an electric drill and immediately examined roentgenologically.

In the illustration the fistula is seen to be free of osteogenesis. On one wall is the projecting bone with the filament of copper wire. The second and third experiments will be continued for many months. In the final report will be presented photomicrographs and a detailed description of the various results. Microscopic examination of slides from the first experiment has revealed a complete absence of bone formation in the fistula.

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Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

THE PARANASAL SINUSES

REVIEW OF THE LITERATURE FOR 1940

SAMUEL SALINGER, M.D.
CHICAGO

The literature for 1940 was not as voluminous as that of previous years, owing doubtless to the curtailment of shipments of periodicals from the Continent. However, it is comforting to note the increase in the material reaching us from Latin America and the tendency among physicians of the neighboring countries toward freer use of journals published in the United States. A closer relationship, to the mutual advantage of physicians of Latin America and North America, could easily be fostered by the addition of abstracts in the respective languages of the countries concerned, at the conclusion of each article. If South American periodicals published abstracts in English (a few already do) and North American periodicals in Spanish and Portuguese, not only wider reading but a deeper interest in each other's activities would result.

ANATOMY

Cave and Haines [1] present an excellent study of the skulls of various types of monkeys, from which certain phylogenetic conclusions may be drawn by comparison with the human skull. This comparison is made graphically in a table, which gives the comparative findings with respect to the sinuses of the gibbon, orang, gorilla, chimpanzee and man. The authors' conclusions may be summed up best in their own words:

> The primitive anthropoid ape stock gave rise to three divergent lines, leading respectively to the relatively primitive gibbons, the highly specialized orang and to the African anthropoid apes and man. Of the last group, the chimpanzee remains a relatively unspecialized member, the gorilla is distinguished by an inflated nasolachrymal duct whilst man is characterized by a remarkably complex ethmoidal labyrinth. The evidence of paranasal structure, considered alone, is inadequate for the demonstration of the mutual relationships of these three forms, yet it does suggest the derivation of both chimpanzee and gorilla from an anthropoid ape common stock distinct from that leading to man. On the other hand the common possession of ethmoidal sinuses suggests a close affinity between man and the African anthropoid apes to the exclusion of all other primates.

1. Cave, A. J. E., and Haines, R. W.: The Paranasal Sinuses of the Anthropoid Ape, J. Anat. **74**:493 (July) 1940.

Maresh [2] studied the sinuses of 100 children by means of roentgenograms taken at intervals from birth to 17 years of age. Superimposed tracings from the films show the method of growth. A number of irregularities were present in different cases, but on the whole the evidence was corroborative of Schaeffer's studies in morphology. The maxillary and ethmoid sinuses seemed to develop more uniformly on both sides than the frontal sinuses. Maresh found that the initial appearance of the latter varied from the second to the eighth year. This was denoted as the time when the sinus reached the level of the root of the orbit. Subsequent studies of this group of subjects may bring out interesting correlative data concerning the bearing of the size and rate of development of the sinuses on the incidence of disease.

Bernfeld's [3] third and concluding contribution on the subject of pneumatization is summed up substantially as follows:

1. The variability of the pneumatization of the sinuses of the nose is the consequence of manifold processes in the development of the bones of the skull.
2. Hereditary factors, in combination with their complicated rules, play the decisive role as to the constancy of the individual differentiation of the various spaces.
3. Inflammation of the nasal mucosa does not exert any particular influence on the degree of pneumatization. This could be shown in different forms of acute and chronic nonspecific as well as in specific infections of the nasal mucosa by both clinical and roentgen studies.
4. The normal process of development of the sinuses, as well as the rhythm of pneumatization, is strongly influenced by dysfunction of the endocrine glands. With certain endocrine disturbances affecting skeletal development there are corresponding changes in the type of pneumatization, paralleling the changes in the bones of the skull.
5. It is therefore very probable that the physiologic variability in pneumatization is influenced by endocrine factors.

Mellinger [4] injected a number of skulls with contrast mediums, to demonstrate the diploetic veins (Breschet) and their connections. He found these veins very thin walled, with no valves and with dilated areas. In most cases they extended over suture lines. The largest and

2. Maresh, M. M.: Paranasal Sinuses from Birth to Late Adolescence, Am.-J. Dis. Child. **60**:55 (July) 1940.

3. Bernfeld, K.: Neue Gesichtspunkte zur Pneumatisationslehre der Nasennebenhöhlen, Pract. oto-rhino-laryng. **2**:337, 1939.

4. Mellinger, W. J.: Venous Circulation as a Factor in Osteomyelitis of the Skull, Ann. Otol., Rhin. & Laryng. **49**:438 (June) 1940.

most extensive of these veins were found in the temporal and frontal regions. Flow of blood may be in either direction, and stagnation may take place readily, thus favoring the spread of an existing infection. The dangerous area in the frontal bone is that where the air cavity of the sinus merges into the cancellous bone. At this point infection may pass readily from the interior of the sinus into the diploetic stricture by way of the venous channels. These diploetic veins communicate with the veins in the dura and meninges on the inside, as well as with those of the scalp on the outside.

In another communication [5] Mellinger presents the results of his anatomic studies of 200 skulls and a number of wet specimens with reference to the canine fossa, particularly as this bears on the Caldwell-Luc and the Denker operation. Since there is a wide variation in the dimensions of the antrum, it is wise before undertaking either of these operations to study both lateral and intraoral roentgenograms. For instance, a thick piriform margin extending backward for some distance makes the Denker procedure rather difficult. The author found that the nerves and blood vessels which course through small bony canals within the wall of the antrum are so variable in arrangement that it is often difficult to predict the degree of disturbance which might ensue from trauma in the operation. He found that the direction of the incision in the soft tissues bears no relationship to the disturbance in the teeth and recommends the Ziegelman inverted T incision as offering the most exposure with the least trauma. The infraorbital foramens are at variable distances below the intraorbital margin (2 to 13 mm.) "but are usually on a level with the attachment of the anterior end of the inferior turbinate bones. The opening is often directed toward the central incisor of the opposite side, a point to remember in attempting to make an injection into the canal." Mellinger recommends the use of transmitted light placed within the antrum through an opening in the nasoantral walls as a valuable means of outlining the location of the network of canals and thus possibly avoiding injury to them.

Liveriero [6] presents an abstract of a long paper read at the thirty-fourth annual congress of the Société Italienne de Laryngologie, Otologie et Rhinologie, covering the anatomy of the sphenoid sinus. The work is based on a study of sections of fetal and adult heads as well as on roentgen and planographic studies. The latter are particularly interesting because the views shown, taken sectionally in the lateral projection, show clearly the relationship of the sphenoid sinus to the surrounding structures. They also show the individual ethmoid cells and their

5. Mellinger, W. J.: The Canine Fossa, Arch. Otolaryng. **31**:930 (June) 1940.

6. Liveriero, E.: Rapport au XXXIV^{e} Congrè de la Société italienne de laryngologie, otologie et rhinologie, Acta oto-laryng. **28**:387, 1940.

junction with the sphenoid sinus and the pterygomaxillary fossa, which is never seen in the usual projections. Among the points brought out by the study are the following: The ostium is about 5 mm. below the level of the cribriform plate and 10 to 15 mm. above the choanal vault. The rostrum is in the midline, its angle constituting the junction of the anterior and inferior walls, about 15 to 20 mm. below the cribriform plate and in the region of the choanae. In cases of anomaly in the relation between the ethmoid and sphenoid sinuses, when retrobulbar neuritis due to disease of a posterior sinus is present, it is advisable to open all of the sinuses. To be sure of having penetrated the sinus, it is well to remember that the floor of the sinus is the choanal vault. The lateral wall of the sphenoid sinus is very delicate and often dehiscent; it often presents deep depressions, and the danger of entering the orbital cavity or damaging the cavernous sinus or carotid artery must be borne in mind. The roof is in relation to the pituitary; the posterior wall, to the veins and arteries of the base of the brain and the pons. The floor may have ridges forming more or less complete septums. Lateral prolongations into the pterygoid and palatal regions are not uncommon and favor retention. The article has some pleasing color drawings that show the relationship of the sinuses to the important vessels in the vicinity.

Lillie and Simonton [7] describe a rare case in which a misplaced ethmoid cell was contained entirely within the frontal sinus, causing blockage and symptoms of pressure. When photographs of the specimen and a microscopic slide were submitted to Dr. Parsons Schaeffer, he pronounced the structure a frontoethmoid cell "which had ballooned forward into the floor of the frontal sinus." When removed, the mass was like a small thin bony-walled cyst lined and covered with thin cuboidal epithelium (not typical) and communicating with the other air spaces by a small opening at its point of attachment.

PHYSIOLOGY

The contribution of Frenckner and Richtner [8] duplicates to a large degree the work of Proetz on the effect of various pathologic conditions and of drugs on ciliary activity. They used ciliated mucosa from living animals and extirpated adenoids, polyps and mucous membrane from human beings. They found the normal ciliary beat to be 160 to 250 per minute. Repeated infection of the nasopharynx seems to arrest ciliary activity in that region. "Ether narcosis as well as injection narcosis with pernoston and urethane [ethyl carbamate] seems to be

7. Lillie, H. I., and Simonton, K. M.: Developmental Extension of an Anterior Ethmoid Cell Within the Frontal Sinus, Arch. Otolaryng. **32**:32 (July) 1940.

8. Frenckner, P., and Richtner, N. G.: Studien über die Zilienbewegung in den oberen Respirationswegen bei Tieren und Menschen unter normalen und pathologischen Verhältnissen, Acta oto-laryng. **28**:215, 1940.

indifferent, but ethyl chloride seems to have a certain arresting effect." Prolonged hypovitaminosis (vitamins A, C and D) in experimental animals retards ciliary activity. An increase or a decrease in the supply of endocrine hormones has no effect. Sections kept in Ringer's solution are not affected, but sodium chloride solution slows the ciliary beat. A weak solution of ephedrine sulfate increases ciliary activity most. A 1 : 1,000 solution of epinephrine hydrochloride does not affect it. Lower dilutions do. A 2 per cent solution of ephedrine sulfate with 5 per cent azosulfamide (disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate) has a stimulating effect. Expectorants and volatile oils slow ciliary activity.

Uddstromer [9] conducted experiments to determine the degree of participation of the turbinates in nasal respiration insofar as they offer resistance to the inhaled air. This has a bearing on therapeutics, surgical as well as nonsurgical, since it is known that the turbinate normally offers a certain amount of resistance to the air column for physiologic purposes before the latter reaches the nasopharynx. Comparing the action of the turbinate with the normal valvelike action of the nares in this same capacity, Uddstromer by taking measurements with a specially constructed apparatus found that the average relationship between the two was as 68:32 (manometric measurement) in favor of the valve action. Wide variations were found in individual cases. He tries to set up a definite physiologic standard but finds that factors inherent in the general economy, such as occupation, environment and vascular and endocrine forces, may easily disturb this relationship.

McGivern [10] has a test for determining the patency of the maxillary ostium. A needle puncturing the sinus through the inferior meatus is connected with tubing leading into a basin of water. Forced blowing of the nose causes bubbles to appear in the basin. The degree of patency or obstruction is determined by whether this occurs from simple forced exhalation or from exhalation with one nostril closed or with both nostrils closed (first, second or third degree). Similarly, the sniffing of forced inspiration should cause fluid to be aspirated into the antrum. The author believes that many resections of the septum fail to give relief because the middle turbinate obstructs the ostium. He advises more frequent removal of this body when his test shows that ventilation of the sinus is impaired.

Lawton and Ross,[11] by exeriments on living animals with the instillation of an 8 per cent solution of mercurochrome, showed that the fluid

9. Uddstromer, M.: L'importance des cornets pour la résistance dans le nez normal, Acta oto-laryng. **28**:364, 1940.

10. McGivern, C. S.: Relation of Nasal Accessory Sinus Ventilation to Tissue and Physiological Morbidity, M. Rec. **151**:174 (March 6) 1940.

11. Lawton, A. H., and Ross, E. I.: Penetration of Perinasal Tissues by Mercurochrome, Arch. Otolaryng. **32**:853 (Nov.) 1940.

penetrated into the sinuses and passed through all the intervening tissues to reach the dura and cortex of the frontal lobes of the brain. The optimum time for this to take place was about two hours. They claim that this transmission must take place by diffusion. The spread through the lymphatics was more rapid. Control experiments on dead animals failed to show similar diffusion; therefore, it is concluded that movement of living fluids is essential for this action. The object of the experiment was to show the possibilities of the activity of drugs that might be used in the nose.

PATHOLOGY AND BACTERIOLOGY

In a study based on 150 specimens taken from the otolaryngologic registry of the American Academy of Ophthalmology and Otolaryngology at the Army Medical Museum, Washington, D. C., Ash [12] was able to divide the cases of sinusitis represented into the following groups: infectious, 75; allergic, 28; mixed, 47. The following tabulation shows the distribution in more detail:

Distribution of Pathologic Types

	Infectious	Allergic	Mixed
Hyperplastic	36	1	7
Polypoid	14	20	24
Fibrous	20	9	15
Ulcerative	6	1	1
Hemorrhagic	8	.	3
Characterized by follicular nodules	16	8	13

A general summary reveals a comparative scarcity of evidence of any suppurative process in the specimens examined as suggested by the infrequence of polymorphonuclear leukocytes, the prominence in chronic sinusitis of reticulum in the stroma and the basement membrane, the frequent appearance of lymphatic nodes and the development of superficial pseudoglands that may become cysts or abscesses. The last-named were observed in 37 specimens. The series shows that hyperplastic changes are observed more frequently than any other pathologic changes in sinusitis of the infectious type and polyps most frequently in that of the allergic type.

Sato's [13] findings are based on studies of tissues removed at autopsy in 87 cases of leprosy (64 of the tubercular and 21 of the neural type). In the cases of leprosy of the tubercular type he found that the septum

12. Ash, J. E.: Sinusitis from the Viewpoint of the General Pathologist, Tr. Am. Acad. Ophth. **44**:304, 1939.

13. Sato, T.: Histopathological Studies on the Leprosy of the Upper Respiratory Tract: The Leprosy of the Nasal and Accessory Sinuses, Tr. Soc. path. jap. **30**:367, 1940.

was involved in over 50 per cent, the turbinates in 66 to 90 per cent and the sinuses in 32 per cent (antrum) and 22 per cent (ethmoids). The characteristic changes seen in the sinuses are a gelatinous degeneration and a leprous infiltrate of either gelatinous or reticulate form. The typical leprous nodules found on the septum and turbinates with breaking down and subsequent atrophy are not present in the sinal mucosa. Grayish white "dew drop" nodules are seen frequently, but on histologic examination the mucosa shows only a gelatinous swelling, with increase of goblet cells, increase of mucous glands and proliferation around the vessels but no typical formation of leprous nodules. In cases of the neural type of leprosy the lesions are predominantly about the lower ends of the septum and middle turbinates, spreading from there to the middle meatus and eventually to the sinuses. Sato believes that the infection of the nose and sinuses takes place by bacterial invasion from the outside and to some degree by way of the blood stream, and he agrees with most other authors that the nose is the chief portal of entry for the infection of the body.

Gillies [14] inclines to the theory that polyps are the result of an inflammatory process, although he qualifies his statement by saying that there are various degrees of inflammation and that in cases in which polyps are present inflammation does not necessarily mean bacterial invasion. He is definitely against the idea that polyps are neoplastic. The possibility is suggested that a filtrable virus is the causative agent. The histologic features of the specimens he examined were fairly constant: There was stratified columnar epithelium, with occasional mucigenic cells; at times there was metaplasia to a squamous cell type, and at times there was even superficial ulceration. Glands were usually scanty and confined to the base. The bulk of the mass was edematous tissue with few blood vessels and varying numbers of inflammatory cells. The latter were mostly plasma cells and were found around the glands and below the epithelium. Polymorphonuclear cells were found mostly below the ulcerated areas and in cases in which there was considerable nasal discharge. Gillies found large numbers of eosinophils even in cases in which the polyps were not associated with asthma or hay fever. There was also evidence of an escape of mucus from the glands into the surrounding connective tissue. He compares this process to myxomatosis of the peritoneal cavity, in which large masses of gelatinous tissue are formed in response to an escape into the peritoneal cavity of mucus from a mucocele of the appendix or a mucinous ovarian cystoma. There was no evidence of disease of the bones to which the polyps were attached, no osteitis or periostitis.

14. Gillies, A. D.: The Histology of Nasal Polypi, M. J. Australia **2**:149 (Aug. 17) 1940.

Som and Kramer [15] report a case of pituitary neoplasm, with spontaneous cerebrospinal rhinorrhea and meningitis, which resulted in death. At autopsy a fistula leading to the olfactory recess was seen in the floor of the anterior fossa. At the nasal end there was a dural polyp, which probably played a role in the intermittent flow and may have acted as the portal of entry for the infection. In another case of rhinorrhea reported, the condition was due to erosion of the posterior wall of the sphenoid sinus by acute osteomyelitis. There was an inflammatory defect in the dura. The authors list the following causes of cerebrospinal rhinorrhea: (1) trauma (external or surgical), (2) neoplastic invasion, (3) suppurative necrosis, (4) long intracranial pressure from a tumor or hydrocephalus and (5), rarely, idiopathic states.

A case of cholesteatoma of the maxillary sinus in a man of 34 is reported by Monteiro.[16] There was a history of two previous operations on the nose. The nasal meatuses were full of foul cheesy material, which was easily removed, revealing granulations. A probe was passed freely from the nose into the antrum. Roentgenograms showed destruction of the external and mesial walls of the antrum. A Caldwell-Luc operation was performed. The antrum, the ethmoid sinuses and the sphenoid sinus formed one large cavity, filled with foul cholesteatoma. The bony floor of the nasal cavity of the same side was eroded. The patient recovered. The author cites various theories as to the genesis of sinal cholesteatoma and believes that metaplasia of the mucosa is the chief factor.

Weldon [17] presents a brief description of the usual types of pathologic change found in various stages of sinal disease. He emphasizes the fact that chronic disease is the result of unresolved acute infection and the importance of care in cases of acute infection, to preserve a normally functioning mucosa which can resist bacterial invasion.

Grove and Farrior [18] made bacteriologic examinations of 365 sinal washings, 130 polyps and membranes removed from the ethmoid and sphenoid sinuses and 108 membranes removed from the maxillary sinuses at operation. They found that 88 per cent of the washings, 80 per cent of the membranes from the ethmoid and sphenoid sinuses and 96 per cent of the antral membranes gave bacterial growth on culture. Staphylococcic infections predominated over streptococcic. The authors believe this proves that hyperplastic sinusitis is primarily of bacterial origin.

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15. Som, M. L., and Kramer, R.: Cerebrospinal Rhinorrhoea: Pathological Findings, Laryngoscope **50**:1167 (Dec.) 1940.

16. Monteiro, A.: Colesteatoma do seio maxilar do esfenoide e das celulas etmoidais, Acta med., Rio de Janeiro **5**:13 (Jan.) 1940.

17. Weldon, W. A.: Applied Pathology of the Paranasal Sinuses, Kentucky M. J. **38**:594 (Dec.) 1940.

18. Grove, R. C., and Farrior, J. B.: Chronic Hyperplastic Sinusitis in Allergic Patients, J. Allergy **11**:271 (March) 1940.

While this seems to rule out allergy as a factor, they state that bacterial allergy is not to be disregarded. These findings are surprising in view of the fact that in the entire group there was such a large proportion of patients with allergic phenomena: 165 with asthma, 25 with hay fever, 12 with vasomotor rhinitis and 7 with urticaria and angioneurotic edema.

DIAGNOSIS

Among the many items set forth by Vernieuwe[19] in his rather lengthy article are the following, which seem worthy of repetition. The fact that a roentgenogram seems to exclude a lesion does not mean that a lesion is not present. Negative roentgen evidence is only a single factor that must be taken into consideration with others in a clinical examination. When one is persuaded that a roentgenogram is taken under good technical conditions, one may conclude that the picture is not at fault. It is the result of certain immutable physical factors. What may be at fault is the interpretation of the picture. Errors in interpretation would be less frequent if otorhinolaryngologists examined roentgenograms from two points of view. If there is a discrepancy between the interpretations of these pictures and the clinical facts, one should resort to other methods of diagnosis, such as the use of contrast mediums, planography and stereoscopy. These procedures are considered only for exceptional cases. Among the errors Vernieuwe points out is failure to recognize the absence of a frontal sinus or the presence of a very small sinus which is only a diverticulum of an ethmoid cell. Varying density of the anterior wall of the frontal sinus may be a source of error. If there is facial neuralgia on the side of greater density, one may be led into error and operate when infection is not present. Too soft rays may cause a shadow in a normal sinus, and rays that are too hard may fail to show a shadow in a diseased sinus. Anatomic differences of the two sides of the face may lead to errors in diagnosis; the superior maxillas on the two sides may vary in thickness. The age of the patient may lead to error. Young people whose teeth have not all erupted have sinuses different from those of adults. Aged patients who have lost their teeth show hypoplasia and lessening in size of the pneumatic spaces. Changes such as these may be more marked on one side, thus leading to error. Especially in young children shadows due to suppuration of the ethmoid sinus may last for six to eight weeks after clinical healing. This should be taken into account when viewing pictures taken within this time. Patients with allergic sinal conditions present a problem, because shadows like those due to infective disease may be present. Observers have commented on bony changes in the ethmoid labyrinth

19. Vernieuwe, J.: Quelques sources d'erreurs dans l'interprétation des images radiographiques des sinus de la face, Rev. de laryng. **61**:130 (March) 1940.

due to pressure absorption of a bony lamina by numerous polyps and hyperostoses in other areas, caused by irritation. The author refers to Schüller's article, pointing out that pictures taken after lavage in cases in which there is atrophy or ozena may show a shadow due to fluid in the antrum or to temporary swelling of the mucosa. (Schüller's article was abstracted in last year's review.)

Pfahler and Vastine [20] are concerned with the early diagnosis of malignant tumors, before distortion due to the invasion of adjacent cavities has taken place. They show by roentgenograms made with contrast mediums how carcinoma frequently differs from benign growths and hyperplasia in the filling defects produced. Atypical irregular defects confined to only one area are to be regarded with suspicion, since benign growths are more likely to be smooth and regular, while hyperplasias, though often irregular, are more diffuse. Bony changes also may be made out in good roentgenograms. Pfahler and Vastine advise the use of numerous projections, including the upright, and also recommend the sphenoid projection, with a film within the mouth, and the Hirtz mento-vertical projection. They point out the drainage of lymph from various areas, such as that from the floor of the antrum, alveoli and palate to the submaxillary nodes and that from the frontal, sphenoid and ethmoid sinuses and the upper part of the antrum to the lateral pharyngeal nodes. Often the latter escape notice and drainage goes on to the deep cervical, the retromandibular and the submaxillary nodes. The authors quote liberally from the literature as to the types of neoplasm found in various locations and the therapy indicated. Emphasis is placed on the importance of always having in mind the possibility of carcinoma when taking roentgenograms of sinuses. To be suspicious is often to be rewarded with an early diagnosis.

Mayer [21] points out that negative roentgen findings may be due to the fact that the technic is not equal to the occasion or perhaps to the absence of secretion at the moment the pictures are taken. He cites 3 cases in which despite negative results from roentgen examination pure pus was recovered from the antrums on irrigation. [Mayer makes no mention of taking pictures with the patient in the upright position, to show a fluid level, a common omission in routine work. S. S.]

Doyle [22] gives the technical details of the standard projections used at Sir Patrick Dun's Hospital, in Dublin, Ireland. A tilt table is used, which enables the operator to take the various projections with the patient in the sitting posture. The standard projections are the Walters,

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20. Pfahler, G. E., and Vastine, J. H.: Roentgen Diagnosis of Cancer of the Accessory Sinuses, Arch. Otolaryng. **31**:561 (April) 1940.

21. Mayer, F. J.: Ein Beitrag zur Bewertung der Roentgendiagnostik bei Erkrankung der Kieferhöhle, Monatschr. f. Ohrenh **73**:651 (Oct.) 1940.

22. Doyle, M.: The Accessory Sinuses, Radiography **6**:150 (Aug.) 1940.

posteroanterior or Granger (at least the picture books like it), the lateral, and the mentovertical for the sphenoid and posterior ethmoid sinuses. The exact details of the patient's position and the position of the table are given, as well as the position of the tube and the time of exposure. The pictures are finally trimmed and bound together with kodak tape, for uniform filing without the use of envelopes. Doyle says that for children roentgenograms are made only of the antrums, because the other sinuses are not sufficiently developed. [This seems strange in view of the fact that the ethmoid and sphenoid and even the frontal sinuses are present at an earlier age. Any of these may be the seat of disease. S. S.]

Schillinger [23] believes that a survey with opaque mediums should be made in cases in which there are: (1) persistent discharge and few symptoms; (2) symptoms which fail to yield to treatment; (3) constitutional involvement, if the sinuses are suspected; (4) asthma and bacterial allergy; (5) recurring respiratory infections; (6) bronchiectasis; (7) conditions for which surgical treatment is contemplated; (8) recurring aural infections; (9) neuralgia of the face; (10) cerebral symptoms possibly of sphenoid origin; (11) optic neuritis and similar conditions, and (12) conditions treated by roentgen rays and other means (to check the results). The procedure is not only for the determination of the thickness of the membrane and the possible presence of growths but also for gaining knowledge about the function of the membrane. He recommends filling the antrum by puncture and the other sinuses by displacement at two different sittings. He gives details of the maneuvers and illustrations of typical findings. Subsequent studies reveal the rapidity, or rate, of emptying, as suggested by Proetz, with deductions as to the effect of pathologic conditions on this. Schillinger makes a point of control study, i. e., comparison of the usual projections with those made after the instilling of contrast mediums. It is often important also to take roentgenograms after irrigation as well as before. He offers a tabulated explanation of the procedure, which clarifies the subject of the maneuvers and the means of obtaining the best results. This is an excellent review, with adequate detail, which makes it practical especially for those who are not sure of their technic or of what to look for.

Cora Eliseht [24] likes the displacement method for diagnosis with contrast mediums but objects to the Proetz position and various modifications hitherto offered, as involving discomfort and the danger of swallowing the fluid. It is hard also to get the exact amount of negative

23. Schillinger, R.: Opaque Survey of the Nasal Sinuses, Radiology **35**: 1 (July) 1940.

24. Cora Eliseht, F.: Radiografía de seno maxilar con sustancias por pontraste. Método por desplazamiento, Prensa méd. argent. **27**:1454 (July 10) 1940.

pressure in every case. He offers a method which he claims is simpler and easier on the patient. The patient sits in a chair and is told that he should practice thoracic breathing through the mouth. His head is then bent forward, so that the chin touches the sternum. Then a rubber catheter is introduced into one side of the nose, while at the same time the nares are compressed against the septum and the patient breathes quietly through the mouth. Attached to the tube is a syringe containing 5 cc. of the contrast medium. After the fluid is instilled slowly into the nasal chamber, the tube is withdrawn and the patient's head turned toward the shoulder, so that the face is horizontal. He is instructed to close his mouth and to try to inhale forcibly; this maneuver causes the negative pressure necessary to draw the fluid into the sinuses. The head is then turned back to the original face-forward position for rest and the same maneuver repeated. It is done four or five times. If the membranes are swollen, the maneuver is done six to eight times. If the rhinologist wishes to have the posterior sinuses filled, the maneuver is the same except that when the head is turned over the shoulder it is rotated so that the face points upward. Cora Eliseht credits Ermiro de Lima with the autosuction idea (abstracted in the review for 1939) but claims the maneuver suggested here as his own.

Grove and Cooke [25] recommend the use of colloidal thorium dioxide (thorotrast) as a contrast medium. They regard this substance as superior to the iodine solutions, because it is less irritating, less toxic and gives a clearer delineation of polyps and of lesions due to degeneration of the membrane even when it is diluted with one or two parts of water. They have used it in 117 patients, with no untoward effects, and cite several illustrative cases. One of the authors (Grove [26]) writes on the same topic in another periodical and mentions the fluid employed as "umbrathor, a colloidal solution containing 25 per cent thorium dioxide."

Sangiovanni [27] recommends the instillation of iodized poppyseed oil by direct puncture of the antrum through the canine fossa with a stout needle directed slightly outward, to avoid the nasoantral wall. He claims that the procedure is not painful and is less dangerous and more certain than inferior meatal puncture or displacement.

Waldapfel [28] cites the case of a child of 11 years who fell on the face. Swelling and hemorrhage resulted. There was a question of

25. Grove, R. C., and Cooke, R. A.: The Use of Thorium Dioxide in Roentgenography of the Sinuses, Am. J. Roentgenol. **44**:680 (Nov.) 1940.

26. Grove, R. C., and Cooke, R. A.: The Use of Colloidol Thorium Dioxide in Roentgenography of the Paranasal Sinuses, Laryngoscope **50**:1165 (Dec.) 1940.

27. Sangiovanni, V.: La radiografia del seno mascellare con mezzi di contrasto, Arch. ital. di otol. **51**:533 (Nov.) 1939.

28. Waldapfel, R.: Is the X-Ray Examination of the Maxillary Sinus Superior to the Simple Transillumination? Laryngoscope **50**:63 (Jan.) 1940.

possible fracture of the superior maxilla. Transillumination showed the right antrum completely dark. Roentgenograms showed both sinuses equally clear, and no fracture was seen. When the swelling subsided, the right antrum became clear on transillumination. Waldapfel concludes that cloudiness on transillumination may be caused by the swelling of soft tissue or by greater thickness of bone and may not be apparent in roentgenograms. He advises the use of both methods in all cases. [I am sure the author does not mean to imply that transillumination can supplant roentgen examination in the diagnosis of fracture. S. S.]

Henner and Urist [29] present a Hartmann cup-shaped ear forceps bent into a curve to pass through an opening made by a large bore Coakley trocar through the inferior meatus. They claim that the instrument is useful in obtaining mucosa from the antrum for microscopic study without subjecting the patient to a major operation. They indicate its usefulness in checking roentgen evidence and in determining whether thickness of the mucosa is due to true hyperplasia or to infiltration with lymphocytes. The instrument might prove helpful in deciding for or against surgical treatment.

SINUSES IN CHILDREN

Mensing's paper [30] is based on a study of the skulls and sinuses of 88 children at autopsy. He first transilluminated the sinuses, then removed the mucosa for histologic examination and finally filled the sinuses with poured metal. He finds that transillumination is more reliable in older children and is more often in agreement with the pathologic condition in cases of empyema and markedly swollen mucosa than in cases of edema with serous exudate. [These conclusions must be discounted because transillumination done on cadavers is far different from that done on the living, on account of the absence of circulation and other postmortem changes. S. S.] Mensing's measurements agree with those of Onodi and Leicher. He also agrees with Von Gilse that the factors necessary for the consummation of pneumatization are a normal mucosa with normal pneumatizing drive and a layer of compact bone bordering on a substantia spongiosa. However, he claims that the pneumatizing force may be slowed down by reason of exogenous influences. This is disputed by authors cited in this and in previous reviews. He also claims that asymmetry was observed in cases in which there had been prolonged infection. Another observation of interest was

29. Henner, R., and Urist, M.: A Forceps for Removing Antral Mucosa for Biopsy, Arch. Otolaryng. **32**:523 (Sept.) 1940.

30. Mensing, G.: Untersuchungen bei Kindern über den Einfluss des Schleimhaut-characters auf die Entwicklung der Nasennebenhöhlen, zugleich ein Beitrag zur Frage des Wertes der Diaphanoskopie, Arch. f. Ohrenh. **146**:440, 1939.

that in many children under 2 years pneumonia was associated with the presence of hyperplastic sinal mucosa infiltrated with numerous lymphocytes.

Benner's studies [31] of a series of 70 stillborn and recently dead newborn infants revealed that 26 per cent of the stillborn and 46 per cent of the liveborn infants who died when under 2 days of age showed evidence of infection of the respiratory tract, ears and sinuses. She believes that aspiration of amniotic fluid is the cause and that the infection occurs after early rupture of the membranes. The parallel conditions seen in the ears, sinuses and lungs seem to indicate a common origin of infection. Recognition of amniotic fluid in these cavities is reliable when cornified epithelial cells and vernix caseosa are present.

McLendon and Rathbone [32] present a fairly complete résumé of their subject, with appropriate references to the literature, the whole constituting an adequate critique so far as causation and pathologic changes are concerned. Their results in the roentgen treatment of 291 patients who were followed for periods of one month to four years showed 60 per cent cures. Of 50 allergic patients followed from one to four years, 39 showed improvement and 11 were cured. These results are rather favorable and are probably due to the cure of an associated infective condition or at least to some shrinking effect on the membrane which lessened the symptoms. In a large number of cases the authors found sinusitis in association with other acute infections of the respiratory tract as well as with allergy.

Mitchell [33] has seen a number of children whose sinuses failed to develop because of neglect of sinusitis. He feels that an undeveloped sinus is usually the seat of chronic disease and, conversely, that when the sinus develops to adult size the infection will disappear. Heredity, functional activity of the organs of internal secretion and local factors, such as diet and environment, all play a part in this process. He agrees with Kaiser that removal of the tonsils and adenoids frequently predisposes to sinusitis. He has found a wide variety of general conditions resulting from sinal disease, the most common being the return of adenoid symptoms,failure to gain weight, recurring bronchitis, pyelitis, asthma and nephritis. A complete survey of the child, including a search for allergens, precedes his outline of treatment, which assumes various forms, as indicated by the conditions observed. Iodides, treatment with heat, change of climate, vitamins, thyroid, calcium, vaccines

31. Benner, M. C.: Congenital Infection of the Lungs, Middle Ears and Nasal Accessory Sinuses, Arch. Path. **29**:455 (April) 1940.

32. McLendon, P. A., and Rathbone, R. R.: Chronic Sinus Infection in Children, South. M. J. **33**:366 (April) 1940.

33. Mitchell, E. C.: The Treatment of Sinusitis in Children, West Virginia M. J. **36**:8 (Jan.) 1940.

and other agents are employed at various times. He also refers to antrostomy as done by Dr. Shea, which has proved helpful in stubborn cases.

Guthrie [34] has found sinusitis prevalent on the south coast of Australia because of high winds with dust and high daytime temperature in summer with a drop of 20 to 30 degrees (F.) at night. Roentgenograms are of great value in the diagnosis. He states that in every case one must treat the patient as well as the sinus and deal with an allergic state if present, and, in the vast majority of cases, use conservative means of treatment. He suggests certain debatable points for future discussion: (1) Should routine lavage of the antrum be done at all operations on the tonsils and adenoids? (2) Is the sinus a focus of infection in arthritis, anemia, nephritis and other diseases in children? (3) What is the best method of general treatment (dietetic, climatic, medicinal, etc)? (4) Of what relative value are local applications? (5) To what extent does roentgen examination help in diagnosis? (6) When is operation justified? Guthrie finds the Proetz method less applicable to children than to adults and prefers to use suction by means of a catheter, and tampons treated with mild protein silver and left in situ for one or two hours. He claims excellent results through these procedures. As for nose drops, he has used 5 per cent gomenol, a preparation of cajuput oil from Melaleuca riridifloral in olive oil, for fifteen years and has never seen any ill effects.

In a study of some 3,000 cases Quillian [35] found that 45 per cent of pediatric practice in southern Florida is concerned with respiratory diseases. He cites the various etiologic factors and the usual methods of diagnosis. Positive roentgen findings plus nasal discharge and enlargement of the cervical glands are presumptive evidence of sinal infection. For treatment he recommends suction for the removal of secretions, the use of ephedrine solutions in saline solution and attention to the general etiologic factors that have contributed to the infection.

Gewanter,[36] after reviewing the anatomy and pathology of the sinuses in children, goes into considerable detail as to diagnosis. He emphasizes the importance of the following symptoms: frequent colds, nasal obstruction, discharge, low grade fever, cough, swelling of the middle turbinates, redness of the lateral pharyngeal bands and the presence of lymphatic nodules in the pharynx. He gives the details of displacement irrigation as first described by Proetz, which he used every two to four days. A 0.5 per cent solution of ephedrine sulfate in saline solution or a 0.125

34. Guthrie, D.: Nasal Sinusitis in Children, Brit. M. J. **1**:84 (Jan. 20) 1940.

35. Quillian, W. W.: Infection of the Nasal Accessory Sinuses in Children, J. Florida M. A. **27**:127, 1940.

36. Gewanter, R.: Conservative Treatment of Sinusitis in Children, Arch. Otolaryng. **32**:728 (Oct.) 1940.

per cent solution of neo-synephrin hydrochloride is used with suction at 180 mm. of mercury eight to ten times on each side. He sees no danger or contraindication to this method of treatment and cites several illustrative cases.

Beck and Fabricant [37] review the literature for the past two years and give excellent summaries of some leading contributions.

D'Eloia [38] finds a parallel between sinal disease and the tuberculosis of childhood in that they rarely present typical symptoms as found in adults and the patients have many manifestations in common, such as pallor, chronic cough, poor appetite, restless sleep and irritability. He says that in children the response to both diseases is cellular, whereas in adults it is inflammatory. Chronic sinobronchitis in children is due to an acute attack, although a patient in whom the disease is chronic may have acute intercurrent attacks. The chief condition observed which leads D'Eloia to call the response "cellular" is hypertrophy of the membranes of the sinuses, nose and nasopharynx. He has had good results from roentgen therapy in these conditions and recommends small doses, long wavelengths and little filtration: 87 kilovolts, 4 milliamperes, 1.5 mm. of aluminum, two minutes' exposure, 12 inches (30.5 cm.) distance and 50 roentgens (r) measured in air. Treatments are given every other day for two weeks. After a week's interval the same course is repeated if indicated.

Poe [39] describes a case of osteomyelitis involving many of the bones of the skull as well as the lacrimal and nasal bones, with metastasis in various parts of the body. The illness came on quickly in a child of 10 years, with headache and, within twenty-four hours, edema of the eyelids, forehead and parietal regions. Roentgen and routine examinations showed pansinusitis, and an operation was performed by the external route, the sinuses being opened and drained. [The extent of the surgical procedure is not mentioned. S. S.] A short time later (the exact period is not noted) there was increased edema over the frontal area, which led to a revision of the wound. This included an attempt to exenterate the frontal and ethmoid sinuses. Four months later metastatic infection appeared. The frontal region did not heal. Fifteen months after the onset there was still a gaping draining wound in the frontal area, with loose sequestrums and scars in the metastatic areas in the radial region of the right arm and a draining fistula in the gluteal

37. Beck, J. C. and Fabricant, N. D.: Sinusitis in Children, Am. J. M. Sc. **199**:865 (June) 1940.

38. D'Eloia, A. S.: Sinus Disease in Children and Its Relation to the Childhood Type of Tuberculosis, M. Rec. **151**:432 (June 19) 1940.

39. Poe, D. L.: Osteomyelitis of Bones of the Head with Multiple Mestastatic Abscesses Following an Acute Attack of Pansinusitis in a Child Followed by Recovery, Arch. Pediat. **57**:422 (July) 1940.

region. The patient was undernourished and nervous. The treatment consisted of irrigations with a bacteriophage and general building up. After six months, the bacteriophage seemed to lose its effect, and potassium permanganate irrigations were used with apparently good effects. Light therapy also was used. The wound eventually healed, and two years later the patient was well and had not had a recurrence. [The important question lies in the relationship of the primary surgical procedure to the subsequent osteomyelitis. S. S.]

Paufique, Sargnon and Bonnamour [40] report the case of an infant in whom three weeks after a difficult instrumental delivery swelling and redness of the area surrounding the right eye developed. After incision and drainage there was a fall in the child's temperature. However, the swelling persisted, and pus was seen coming from the region of the middle turbinate. Since the greater part of the swelling was below the eye rather than above it, the authors believed that they were dealing with infantile osteomyelitis of the superior maxilla. They instituted drainage through a large rubber tube and used collubiazol [40a] for irrigation. Within two days the discharge diminished, but diarrhea appeared, which they thought might be due to aspiration of the irrigation fluid, the administration of which was then discontinued. The temperature gradually subsided, and at the same time a metastatic abscess appeared over the sternum. This was incised and drained. Later roentgenograms confirmed the diagnosis of the maxillary origin of the infection. Several months later the child was reported well. [There is a question as to the diagnosis, since it is known that conditions such as these usually are accompanied by marked prostration and that when they last long enough, sequestrums always appear. The roentgenograms submitted are not conclusive. S.S.]

Piaget reports a case [41] of orbital phlegmon in a 2 month old infant. The condition developed in a few hours after the onset of coryza. The classic symptoms of chemosis, edema, papillary stasis and external swelling were present. Relief was obtained by external incision. Piaget comments on the extreme youth of the patient. Since at birth there are not more than two or three small ethmoid cells, it is rare indeed that their inflammation leads to abscess of the orbit. He quoted Envard, who

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40. Paufique, Sargnon and Bonnamour: Osteomyélite aiguë du maxillaire supérieur et phlegmon de l'orbite droit chez un enfant de trois semaines, drainage orbitaire, guérison complète, Ann. d'oto-laryng., September-October 1939, p. 851.

40a. This is a foreign proprietary preparation. The article cited does not give any information about it, and the Council on Pharmacy and Chemistry of the American Medical Association has no record of it.

41. Piaget: Ethmoïdite avec phlegmon de l'orbite chez un enfant de deux mois; traitement chirurgical, guérison, Ann. d'oto-laryng., September-October 1939, p. 848.

reported on 50 patients with orbital complications, the youngest of whom was 19 months old.

Hall [42] presents a general review of sinal disease in children, with references to the literature. He believes that many cases are overlooked because the symptoms are so variable and not characteristic except when taken in conjunction with the contributing factors. He considers it important that children with sinal disease be followed up for some time and checked with occasional roentgenograms after apparent recovery.

INTRACRANIAL COMPLICATIONS

Meningitis.—Neal, Appelbaum and Jackson [43] report a series of 30 cases of pneumococcic meningitis, with 10 recoveries, and 29 cases of meningitis due to Bacillus influenzae, with 14 recoveries. Treatment was with serum and sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine). The authors attribute the large percentage of recoveries as compared with those recorded in the old time reports to the effectiveness of this drug. Only 1 instance of pneumococcic meningitis and 2 of meningitis caused by B. influenzae had their source in sinal infection. All 3 patients recovered. It is to be noted that the drug was used intraspinally.

Kramer and Som [44] present serial sections from autopsy specimens in a number of cases of meningitis. These reveal the details of the pathways of infection from the ethmoid and sphenoid sinuses. The spread to the meninges occurred by osteitis and osteomyelitis, through lymphatic channels and venous channels, directly through bony defects and from a persistent cranial pouch. Kramer and Som frequently found a small submucous abscess in the sphenoid and ethmoid sinuses, which suggests that in cases of meningeal irritation if the sinuses are involved the mucosa should be stripped for better drainage. Often there were few or no gross changes in the sinuses, but microscopic studies revealed connecting pathologic conditions. Only 3 of 50 blocks studied showed gross changes. This proves that a lesion does not have to be massive to provoke serious consequences.

Pallestrini [45] claims that the percentage of recoveries from meningitis has risen from between 5 and 9 per cent to 33.3 per cent since the advent of chemotherapy. He cites the cases of 8 patients who under his care

42. Hall, C. H.: Sinus Disease in Children, J. Oklahoma M. A. **33**:12 (Nov.) 1940.

43. Neal, J. B.; Appelbaum, E., and Jackson, H. W.: Sulfapyridine and Its Sodium Salt in the Treatment of Meningitis Due to the Pneumococcus and Haemophilus Influenzae, J. A. M. A. **115**:2055 (Dec. 14) 1940.

44. Kramer, R., and Som, M. L.: Intracranial Pathways of Infection from Diseases of the Sphenoid and Ethmoid Sinuses, Arch. Otolaryng. **32**:744 (Oct.) 1940.

45. Pallestrini, E.: Sul trattamento delle meningitis purulent otogene e rinogene, Accad. med., Genova **54**:596 (July) 1939.

recovered through combined surgical intervention and chemotherapy. One of these had acute purulent frontal sinusitis with circumscribed osteitis and leptomeningitis. The spinal fluid was turbid, with large numbers of cells, both polymorphonuclears and lymphocytes, and gram-negative extracellular diplococci. Recovery followed extensive surgical treatment plus large doses of azosulfamide (disodium 4-sulfamidophenyl-2-azo-7′-acetylamino-1′-hydronaphthalene-3′-.6′-disulfonate).

Encephalitis.—Yaskin [46] discusses the difficulties in diagnosing certain intracranial complications during life, because of the similarity of symptoms and frequent lack of pathognomonic signs. For this reason and because the lesion is rarely diagnosed in life, localized encephalitis has been recorded in the literature infrequently. The author states that the condition is the same as brain abscess except that suppuration fails to take place. In most of the cases reported hitherto the encephalitis has resulted from infection of the temporal bone. Only Behlu and Atkinson have reported cases in which the condition arose from sinal disease. Among 4 cases studied by Yaskin was 1 of this type. The patient, a man of 28, had a chronic sinal infection and was operated on some years before. A month prior to admission he had acute sinusitis, which seemed to abate for a while. About ten days prior to admission there were recurrent headaches. Later there appeared twitching of the left side of the face with severe frontotemporal headache. The patient then lost consciousness for fifteen minutes and on coming to vomited. His temperature was 100 F. On examination the left ocular disk was elevated and there were central palsy of the facial nerve on the left, impairment of the sense of position and astereognosis in the left hand. For several days the temperature was nearly normal. Roentgen examination showed normal conditions of the skull and involvement of the frontal, ethmoid and maxillary sinuses. For several days the eyegrounds showed persistent elevation of the disks. In two days after admission the sensory changes disappeared, but the palsy of the facial nerve persisted. The headaches decreased with repeated lumbar punctures and local nasal treatment. (The spinal fluid earlier showed a pressure of 300 mm. of water with 150 cells, mostly lymphocytes, and protein. This cleared up rapidly after subsequent taps.) Subsidence of focal signs prevented exploration for a suspected abscess. Yaskin points out how the contralateral side of the brain was affected from the sinal infection. Analyzing the symptoms and course in cases such as this, he directs attention to the importance of being on the alert in order to avoid too early intervention in the belief that an abscess is present and also to the importance of early eradication of the etiologic focus.

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46. Yaskin, J. C.: Localized Nonsuppurative Encephalitis Secondary to Infections of the Temporal Bone and Paranasal Sinuses, J. Nerv. & Ment. Dis. **92**:281 (Sept.) 1940.

Rouquier [47] cites several cases of subacute and chronic maxillary and frontal sinusitis in which he was able to demonstrate spontaneous deviation of the arms to the side of the lesion and a tendency to fall to the same side when walking with the eyes closed. The galvanic test also showed more marked reactions to the affected side. In some cases there was no vertigo. Migrainous headache was frequently encountered. The author feels that the condition is due to irritation in the subarachnoid spaces or the dura, causing a reaction in the cortex which affects the frontocerebellar pathways.

Embolus.—Brunetti [48] describes a case in which a tumor was removed from the maxillary and ethmoid sinuses through a Denker approach. The tumor presented characteristics of a lymphoepithelioma, or Schmincke's tumor, although the pathologist thought it might also be called a lymphosarcoma. At any rate, the characteristic cell type was lymphoid. Thirty-six hours after operation the patient suddenly had an attack of extreme restlessness, with a sense of feeling very ill. This was followed by loss of consciousness. When, soon afterward, the patient recovered consciousness, aphasia was present. This was followed by symptoms of meningitis leading to a fatal outcome. Autopsy revealed meningitis, with pneumococci in the spinal fluid. Brunetti believes that the sudden onset of symptoms plus the aphasia can be explained only on the basis of an embolus obstructing the circulation in the fissure of Sylvius, although this could not be determined definitely at the autopsy.

Thrombosis of the Cavernous Sinus.—Rubin and Cutler [49] report a case of thrombosis of the cavernous sinus due to exacerbation of a chronic sinal disease (sphenoid-ethmoid). The patient lived one week under treatment with heavy doses of sulfanilamide and spinal taps. Autopsy revealed pus and destruction of bone in the ethmoid and sphenoid sinuses, generalized meningitis and thrombosis of the cavernous sinus and tributary veins. In another case also, that of a youth of 18, the condition was due to fulminating acute sphenoethmoid disease. A significant symptom pointed out by the authors was the presence of severe occipital headache for a week before the cerebral symptoms appeared. This symptom evidently had been minimized, the condition being treated as influenza. The authors point to the difficulty of diagnosing an infection of the sphenoid sinus, especially if the patient is seen at home.

47. Rouquier, A.: L'encéphalite frontale; complication presque constante des sinusitis aiguës ou chroniques, Ann. d'oto-laryng., November-December 1939, p. 898.

48. Brunetti, F.: Tumore dei seni facciale di S. sintomatologia di embolo cerebrale; exitus, Gior. ven. di sc. med. **14**:129 (March) 1940.

49. Rubin, H. W., and Cutler, A.: Cavernous Sinus Thrombosis: A Report of Three Cases with Autopsy Findings, Ann. Otol., Rhin. & Laryng. **49**:736 (Sept.) 1940.

SINUSES IN RELATION TO DISEASES OF OTHER ORGANS

Heitger [50] presents a general discussion of the relationship of sinal disease to arthritis, mental disorders and other diseases in regions more or less remote, with a number of references to the literature. He emphasizes the necessity for accurate appraisal of the focus and the importance of being able to diagnose the latent types of disease, the elimination of which often leads to startling results.

Pulmonary Disease.—Goodale [51] presents a series of 150 cases of bronchiectasis, 76 of which were studied to illustrate the relation of disease of the sinuses to infections of the chest. Lobectomy was done on 21 patients whose sinuses were normal; of these, 8 became symptom free, 8 were markedly improved and 5 were moderately improved. Fourteen patients with sinal disease had lobectomy, and of these 10 showed marked improvement. On 11 other patients both lobectomy and operations on the sinuses were performed; 7 showed marked improvement, 2 moderate improvement and 2 slight improvement. Of 30 patients who had operations on the sinuses alone, 3 were symptom free, 13 markedly improved, 4 slightly improved and 10 unaffected. Since the connection between the two conditions is not always clear and since the degree of chronicity has much to do with the case, it is apparent that each case has to be handled on its own merits. The best results from lobectomy were obtained in those cases in which there was no sinal disease. Patients for whom lobectomy was not indicated showed improvement after surgical treatment of the sinuses. However, this has not proved an adequate substitute for lobectomy when the latter has been indicated.

From a résumé of the literature and his own observations Smith [52] reports that there is a strong prenatal developmental factor in the incidence of respiratory disease, particularly bronchial dilatation. He refers to the established routes of transmission of infection from the nasal passages to the lower air tract as etiologic factors in the development of many of the infections in the latter region. Infection produces the same surface and deep reactions in the upper and in the lower respiratory tracts. These may occur at the same time. He believes that the factors of allergy and diet have not been definitely established. He concedes the influence of hormones. Mechanical obstruction and infection are, in his opinion, the essential factors in the production of bronchiectasis.

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50. Heitger, J. D.: Sinus Disease in Relation to Systemic Disease, Kentucky M. J. **38**:285 (July) 1940.

51. Goodale, R. L.: The Relation of Sinusitis to Chronic Non-Tuberculous Chest Infection, New England J. Med. **223**:654 (Oct. 24) 1940.

52. Smith, F.: Correlation of Chronic Infection of the Upper and Lower Respiratory Tracts, Pennsylvania M. J. **43**:1389 (July) 1940.

An interesting case reported by Cole and Nalls [53] concerns a man of 22 whose complaint was of headache and impairment of hearing, accompanied with chronic bronchitis since childhood. Sinusitis was diagnosed when he was 9 years of age, because of headaches. The antrums were opened and drained, with temporary relief. Recurring nasal polyps were removed several times. In 1930 radium treatment was applied to the nose, without benefit. Later an operation on the septum was done, and still later, a radical operation on the antrum. In 1937 a series of roentgen treatments of the sinuses was given, without benefit. The sense of smell was gone, and the nasal discharge persisted. There had been chronic cough and expectoration since childhood. In April 1940 the patient had atypical pneumonia, and at this time he was told that his heart was on the right side. Examination revealed a pale nasal mucosa and much mucus in both sides of the nose. Roentgenograms showed, in addition to pansinusitis, the heart on the right side, the stomach on the right side and bilateral bronchiectasis, with thickening of the bronchial walls.

Nephritis.—Abbate [54] cites a case of chronic nephritis with hypertension in a woman of 25 who was suffering from pansinusitis (suppurative) on one side. Cure of the sinal condition brought relief from the nephritis and hypertension. Abbate reviews the whole subject of focal infection so far as the sinuses are concerned and concludes that the kidneys may be affected more often than is believed, by a focus in the sinus which may be overlooked.

Deafness.—Jordan [55] refers to an article by Whiteman [56] (abstracted by me in the review published two years ago [57]) in which it was claimed that in certain cases deafness and tinnitus were greatly benefited by irrigation of the antrums. The procedure consisted of prolonged inhalations of menthol vapor by the patients at home and surgical antrostomy, with subsequent daily irrigation of the antrums with physiologic solution of sodium chloride. At the same time the patient used a menthol spray frequently or placed a tampon saturated with a solution of menthol in the naris for inhalation several times a day. Jordan cites several cases,

53. Cole, D. B., and Nalls, W. L.: Situs Inversus, Sinusitis and Bronchiectasis, J. Thoracic Surg. **9**:689 (Aug.) 1940.

54. Abbate, L.: Nefrite cronica ipertensiva da sinusite etmoido-sfenoidale cronica, Oto-rino-laring. ital. **10**:74 (Jan.) 1940.

55. Jordan, R. R.: Sorderas y zumbidos; el exito en ciertos casos del moderno tratamiento australiano, Rev. méd. del Rosario **30**:665 (June) 1940.

56. Whiteman, R. J.: Neue Therapie für Schwerhörige, Wien. med. Wchnschr. **87**:900 (Aug. 28) 1937.

57. Salinger, S.: Paranasal Sinuses: Summaries of Bibliographic Material Available in the Field of Otolaryngology, Arch. Otolaryng. **28**:252-293 (Aug.) 1938.

chiefly cases of tubal catarrh in which the patients were cured or greatly benefited by the treatment. He concludes that the procedure is indicated in cases in which tubal catarrh is aggravated by chronic nasal catarrh. He states also that he has seen good results even in cases of undoubted otosclerosis, for which he can offer no explanation.

Osteomyelitis.—Bucy and Haverfield [58] report 3 cases of osteomyelitis following swimming and 1 in which the disease was due to trauma to a stye. The case histories make interesting reading, and the gratifying results are a tribute to intelligent management and recognition of the disease. Bucy and Haverfield are convinced that the infection spreads by way of the venous system and believe that prompt external exposure, with wide excision beyond the affected area, is the logical way to prevent further spread of the process and insure recovery. When brain abscess is present, they advocate opening it through an uninfected area, with proper preliminary walling off of the meninges. They believe that the bony defects remaining after wide excision are ultimately repaired by regeneration of the bone.

Kettel [59] is convinced of the necessity of adequate exposure in order to remove all of the diseased tissue properly. He recommends the coronal incision above the hair line, from ear to ear through the scalp and periosteum down to the bone, with reflection of the flap forward. There is little bleeding, because no large vessels are encountered. With this exposure of the entire frontal and part of the parietal areas it is easy to remove all of the diseased bone, treat the dural and subdural spaces and exenterate the affected sinuses. The cavity is drained into the nose, and the flap is then replaced and partly sutured. Small drains are placed in the angles, and these are kept soaked in a solution of sodium hypochlorite. When the secretion lessens, the scalp can be sutured in place completely. The scar, being within the hair line, is rarely visible. Kettel states that Adson and Hempstead have described the same procedure independently. [Mygind also described the procedure. S. S.]

Jones [60] believes that many physicians are still unmindful of the possibility of osteomyelitis, often overlooking this process until it is too late to effect a cure. He cites several experiences to prove this. Recently he has used Furstenberg's hair line incision, as it leaves less scar than the T-shaped incision he formerly used. In 7 of his cases the osteomyelitis was of the acute, subacute or chronic type, but in none was

58. Bucy, P. C. and Haverfield, W. T.: Cranial and Intracranial Complications of Acute Frontal Sinusitis, J. A. M. A. **115**:983 (Sept. 21) 1940.

59. Kettel, K.: Osteomyelitis of the Frontal Bone, Arch. Otolaryng. **31**:622 (April) 1940.

60. Jones, A. C.: Osteomyelitis of the Frontal Bone with Report of Thirteen Cases, Ann. Otol., Rhin. & Laryng. **49**:713 (Sept.) 1940.

it fulminating, and all the patients got well. In 2 cases the bone disease followed trauma. In the first of these the patient was a child of 2½ years. There was a history of trauma to the head nine weeks previously. The disease ran a chronic course for several months. Operation was followed by recurrence finally leading to brain abscess and death. The second case was that of a woman of 56 with a history of trauma three years previously. She had paralysis of the face and arm on the left side and chronic osteomyelitis. In the course of operation the dura was torn, with escape of fluid. The patient recovered. There were 2 cases of osteomyelitis with cerebral complications. In the first of these the condition was due to swimming. Gas was present in the pus, due to carbonic acid produced by the organism, and there was localized meningitis. Recovery was slow. In the other case also the condition followed swimming and was acute and severe. Operation was followed by symptoms of cortical irritation. Subsequently the brain was explored and bloody fluid was released from the frontal lobe. Recovery ensued.

Porto [61] reports 2 cases in which osteomyelitis followed a Caldwell-Luc operation within a week. The symptoms were pain, fever, swelling and suppuration in the cheek. The pus was released by incision (gingival or sublabial recess). Extension occurred along the ascending process of the superior maxilla to the frontal bone, with swelling of the lids and later the classic signs of osteomyelitis of the frontal bone. Thence the lesion spread to other cranial bones. In both cases the disease resulted fatally—in one with symptoms of meningitis, in the other with brain abscess. Porto found only 11 cases of this type recorded in the literature. In his opinion, extension from the superior maxilla cranially takes place via the venous channels or the haversian canals. Extensive surgical treatment of the superior maxilla is not feasible or as practical as such treatment of the frontal or other cranial bones, and therefore the lesion cannot be excised completely. Porto states that in the cases reported in the literature the disease was also fatal. He cannot explain the presence of this complication, because it has occurred at the hands of competent surgeons, among others. [It would be interesting to know whether a rasp or sharp curet was used in these cases, since it is known that trauma to the diploetic bone may be the means of implanting the infection. S. S.]

MALIGNANT TUMORS

Robin [62] presents a review of the present status of malignant disease of the maxilla, including reports from the literature and quotations from

61. Porto, G.: Osteo-mielite do cranio consequente a operacâo de Caldwell-Luc, Rev. brasil. de oto-rino-laring. **8**:165 (May-June) 1940.

62. Robin, I. J.: Malignant Disease of the Maxilla, Guy's Hosp. Rep. **89**: 301, 1939.

various authorities. He reports 44 cases encountered at Guy's Hospital, London, England, from 1928 to 1937. They are classified as follows: carcinoma of the antrum, 33; carcinoma of the maxilla, 7; sarcoma of the antrum, 3; sarcoma of the maxilla, 1; carcinoma of the hard palate, 13; carcinoma of the ethmoid cells, 4; carcinoma of the lacrimal sac and ethmoid cells, 1. Various methods of treatment were employed, but a statistical analysis of the results is not completely available. He says it was impossible to trace most of the patients. Typical cases are given in detail. Most of the patients were treated by radical resection, sometimes with diathermy. This was followed by high voltage roentgen therapy. He believes the best results are to be obtained by radical resection including the palate and by subsequent roentgen treatment. He also thinks more patients will receive preoperative treatment with radiation in the future.

Brunner and Wall [63] describe a rare case of metastatic carcinomatosis of the nasal mucosa from carcinoma of the stomach. Death was due to hemorrhage following puncture of the antrum. The more recent symptoms were fulness of the cheek, paralysis of the external oculomotor nerve, exophthalmos and clouding of the antrum, which led to the diagnostic puncture; the naso-antral wall was soft and suggested the possibility of a malignant condition. Autopsy revealed carcinoma of the stomach, with metastases to other abdominal organs, and carcinomatous thrombi of dural vessels as well as of those throughout the nasal mucosa. The interesting finding of emboli in the veins and lymphatics of the nasal mucosa, consisting of irregular-shaped cells with large nuclei and numerous mitoses, is commented on. These cells are identified with those found in the carcinoma of the stomach, from which they unquestionably migrated. The absence of tumorlike formations in the nose made the diagnosis difficult. The similarity of the cells to the onkocytes described by Hamperl [64] and to the cells found in the mucous glands of this case is also commented on. The fatal gastric hemorrhage after puncture of the nasal antrum is analyzed on the basis of a reflex loading of the blood vessels of the region of the stomach. Several authors who have made experimental studies on these lines are quoted freely.

Del Regato [65] discusses the results in 10 cases of carcinoma of the maxillary sinus encountered at the Marie Sklodovsk-Curie Institute of Radium, Warsaw, Poland, cases in which treatment was by irradiation

63. Brunner, H., and Wall, J. W.: Carcinomatosis of the Nasal Mucous Membrane: Fatal Hemorrhage After Puncture of Maxillary Sinus, Ann. Otol., Rhin. & Laryng. **49**:688 (Sept.) 1940.

64. Hamperl: Ueber das Vorkommen von Onkocyten in verschiedenen Organen, Virchows Arch. f. path. Anat. **289**:326, 1937.

65. del Regato, J. A.: Sur la roentgenthérapie des épitheliomas du sinus maxillaire, Radiophys. et radiothérapie **3**:557, 1939.

only. In each of these cases, for some reason or other, surgical intervention was not indicated. Four of the 10 patients were living and well for five years or more. The author claims that in most of the instances carcinoma of the antrum was an epidermoid epithelioma and radiosensitive. The best results were obtained from treatment prolonged five to six weeks. Lesions encroaching on the orbit may endanger the eye, with loss of vision and other injury. When the rays must reach the orbit, he recommends giving up to 2,500 r without protection and then interposing a lead screen 20 to 25 cm. from the eye, to deflect the rays. In the last part of the treatment he places a lead cup directly over the eye. The average total dose in five to six weeks was 6,000 to 7,000 r over two fields. Two of the cured patients lost the vision in the affected eye. Regato states that in those patients who recovered the lesion, while apparently extensive, evidently had not progressed to the point where the orbit or the base of the skull was invaded.

Woodward and Archer [66] present results from irradiation of advanced carcinomas of the sinuses. The results were disappointing. Of 9 patients with epidermoid carcinoma thus treated, all died, the longest survival being thirteen months. The authors feel that advanced carcinoma should be treated surgically, with irradiation only of cervical metastases. Transitional cell carcinoma of the sinuses does well under radiation therapy, but the eye must be sacrificed, because in order to prevent metastases to the brain the base of the skull must also be treated.

Nakamura [67] reports an unusual case, that of a man of 28 who had a tumor of the upper part of the right jaw, developing over a period of two months, with nasal block and epistaxis. The antrum was opened and was found filled with bleeding granulation masses, which extended into the nasal cavity with partial destruction of the nasoantral wall. Histologically the tumor was exceptional because of the atypical giant cells found in a typical fibroblastic sarcoma. The tumor recurred but disappeared again after intense irradiation.

Bajkay [68] states that sarcoma of the nose and paranasal cavities is uncommon. He could find in the medical literature only 590 cases reported, to which he adds 1 of his own. The relatively rare occurrence of nasal and paranasal sarcoma is indicated by the following data: In Gurlt's statistics there were only 15 tumors diagnosed as sarcoma among

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66. Woodward, F. D., and Archer, V. W.: The Results of Treatment of Malignant Tumors of the Larynx, Hypopharynx, Nasopharynx and Sinuses, Virginia M. Monthly **67**:751 (Dec) 1940.

67. Nakamura, M.: Ein Fall von Fibroblastensarkom mit atypischen Riesenzellen in der Kieferhöhle, Bull. Nav. M. A., Japan (Abstr. Sect.) **29**:29 (May 15) 1940.

68. Bajkay, T.: Sarcoma of Unusual Size of Maxillary Sinus, Gyógyászat **80**:31 (Aug.) 1940.

11,000 patients. Herzfeld found 3 among 28,000 patients, and H'Ajutula found 6 among 2,500 patients. According to Schmidt's and Finder's earlier statistics, 0.026 and 0.036 per cent, respectively, of the cases are instances of sarcoma. Fibiger and Trier observed only a single case of paranasal sarcoma among 73 cases of sarcoma. Sarcoma of the nasal cavity most frequently develops in the anterior part of the nasal septum and sarcoma of the paranasal cavities, as Geschelin stated, in the maxillary sinus. In many cases in which the disease is in an advanced stage, there is no longer a chance to detect the primary origin of the tumor and often it is impossible even to determine which paranasal cavity was primarily affected by the growth. Sarcoma of the nasal cavity renders breathing through the nose first difficult, later even impossible. Twang, headache, olfactory disorders, tinnitus, diminished hearing and earache are the outstanding symptoms. Bleeding from the nose, either spontaneous or following trauma, belongs to the early symptoms; later, disintegration of the tumor and erosion of the vessels cause hemorrhage. Further pathologic developments may include deformity of the nose, deflection and perforation of the septum, a foul purulent discharge, a positive finding by posterior rhinoscopy, spreading of the process to the face, paralysis of the optic muscles, exophthalmos, disturbances of vision, atrophy of the optic nerve, increased intracranial pressure, meningitis, extradural abscess, brain abscess and thrombosis of the cavernous sinus. Tumor arising from a paranasal cavity may cause severe headache and neuralgia at an early stage, the first branch of the fifth nerve being involved in tumor of the ethmoid sinus. On the other hand, the second branch of the same nerve is affected in tumor of the sphenoid sinus. Each of these tumors may grow into the neighboring cavities as well as into the orbit, causing characteristic symptoms. Metastasis to the submaxillary lymph nodes is relatively rare, because the nasal lymph vessels empty into the subarachnoid and the subdural spaces. Prognosis depends on early diagnosis and early removal of the tumor and on the site, size and microscopic structure of the growth, as well as on the general condition and age of the patient. Bajkay cites cases from the literature in which operation has been successful, with no recurrence for periods as long as ten to fourteen years. The patient reported on in the paper reviewed here lived for two years after the operation in spite of the unusual size of the sarcoma (97 Gm.) and died of intercurrent disease. The author emphasizes further the importance of early and radical operation followed by roentgen treatment.

Cook [69] states that the best treatment for cancer of the antrum is resection of the superior maxilla according to the time-honored technic.

69. Cook, T. E.: The Surgery of Malignancy of the Maxillary Antrum, Dallas M. J. **26**:130 (Nov.) 1940.

He advises colonic ether-oil anesthesia and temporary compression of the external carotid artery by ligature, for hemostasis. If the tumor extends beyond the confines of the maxilla, he advises subsequent electrodiathermy and irradiation of the involved region. No data in the form of case reports or postoperative results are given. [It is the general opinion of authorities in the largest clinics of the world that radical extirpation of the maxilla is not as effective as removal of the tumor by surgical diathermy, followed by radiation therapy. S. S.]

Breeding [70] reports 2 cases.

The patient in the first case had headaches and pain in the eye and frontal sinus for two months, with tenderness and nasal discharge. A diagnosis of frontoethmoid and antral disease was made, and operation carried out by making bilateral antral window and intranasal ethmoidectomy. One month later the pain and tenderness over the frontal sinus had increased, and there was some swelling. An external operation disclosed erosion of the anterior wall of the frontal sinus as well as of the posterior wall, exposing the dura. Large masses of tumor were found attached to the bone, periosteum and dura, which on histologic examination proved to be squamous cell carcinoma. Roentgen treatment gave only palliative results. In the second case a man of 62 presented himself with pain, swelling over the frontal sinus and a dense cystic mass resembling a mucocele in the frontoethmoid. Roentgen examination showed absorption of bone. An external operation revealed hard cystic masses filling the entire frontal sinus and extending into the ethmoid region. Histologically the growth was transitional cell carcinoma. It recurred after roentgen therapy.

Brownell [71] reports a case which illustrates the danger that a benign tumor of the jaw may become malignant. A chondroma of the alveolus in a girl of 13 invaded the anterior wall of the antrum. It was resected and shown to have areas of sarcoma. There were recurrences and several subsequent resections. The rapid growth of the neoplasm ultimately proved fatal to the patient, the mass having reached enormous proportions. Autopsy showed the tumor to be a spindle cell fibrosarcoma. Biopsy of material removed at the first operation resulted in a diagnosis of "chondrosarcoma." The second case was that of a 15 year old Negro boy who had swelling of the cheek for two years. Shortly before examination the growth became more rapid. The tumor involved the antral region, pushing the nasoantral wall medially. A large firm mass caused

70. Breeding, E. G.: Malignancy of the Nasal Accessory Sinuses with a Report of Two Cases of Primary Carcinoma of the Frontal Sinuses, Ann. Otol., Rhin. & Laryng. **49**:141 (March) 1940.

71. Brownell, D. W.: Chondroma and Fibroma of the Superior Maxilla: Report of Two Cases, Ann. Otol., Rhin. & Laryng. **49**:765 (Sept.) 1940.

swelling of the cheek extending to the orbital margin and alveolus. The report of a biopsy was: "Cellular fibroma with suspicion of sarcomatous involvement." Operation showed that the tumor had eroded the mesial wall of the antrum, the roof, the posterior wall, the zygoma and hard palate. Histologic examination revealed tissue varying from dense fibroma to spindle cell osteochondrosarcoma. Radium therapy was followed by a temporary recession and then a recurrence two months later. This tumor was removed, and a cure seemed in effect. A tube flap was raised to close the defect, but the tumor recurred again with fatal result.

NONMALIGNANT TUMORS

Osteoma.—In seven years at the Kasr-el-Ainy Hospital, in Cairo, Egypt, among 840,000 patients treated in the ear, nose and throat department Handousa [72] found only 37 with benign tumor of the nasal region, in 18 of whom the growth was osteoma. Several good photographs and roentgenograms make the article interesting. Analysis shows that the majority of the patients in whom osteoma was discovered were below the age of 28. This plus the fact that the tumor takes a long time to reach its full growth and then ceases to grow leads him to the conclusion that osteoma in this region, like osteoma of a long bone, is a disease of the bones during their period of growth. Most of the neoplasms were single, and there were no instances of associated osteoma in other parts of the body. In the first 13 cases seen the osteoma originated from the frontal bone. Handousa shows that in all instances it originated in the region of one of the epiphyses of the frontal bone. This conforms to the rule which he cites from Choyce's "A System of Surgery": [72a] "The position in which a spongy osteoma arises from a bone is very constantly the diaphysis in the immediate neighborhood of an epiphysial line, although as the growth of bone advances, the distance between the tumor and the epiphysis is proportionately increased." The author points out the location of centers of ossification in the frontal and ethmoid sinuses and shows their relationship to tumors he found. He explains the presence of cancellous osteoma of the superior maxilla in 2 cases. A neoplasm of this type is supposed to develop in membrane, so that one would expect the tumor to be compact. In this connection he writes, "But according to Frazer,[72b] there may be a cartilaginous change here and there in the alveolar condensation of the superior maxilla—so obviating this

72. Handousa, A. S.: Nasal Osteomata, J. Laryng. & Otol. **55**:197 (April) 1940.

72a. Johnson, R., in Choyce, C. C.: A System of Surgery, ed. 2, New York, Paul B. Hoeber, 1923, vol. 1, p. 421.

72b. Frazer, J. E.: Anatomy of the Human Skeleton, Philadelphia, P. Blakiston's Son & Co., 1914, p. 223.

difficulty." He discards the inflammatory and the traumatic theory in favor of the "sequestrated rest" theory, because of the

> almost constant position of the tumour in relation to the epiphysis shown previously in discussing their origin, and by the fact that those osteomata are essentially an affection of the bones during their period of growth . . .
>
> The "sequestrated rest" is supposed to have been separated from the Epiphysis by trauma. This cannot be definitely proved, as the trauma may take place in infancy and cannot be remembered by the patient.

Only 1 of the patients had had a definite trauma at the site of the tumor.

Smith [73] observes that in cases coming under his observation it was difficult to define the limits of osseous growths of the nose and sinuses microscopically. This raises some doubt as to whether the growths were true neoplasms. He cites authors who claim that trauma is the inciting factor but points out also the infrequency of osseous tumors, considering the frequency with which children experience trauma. He observes that the most frequent sites of osteoma are near centers of ossification of cartilaginous bone. Analysis shows that there is a suggestive similarity in the influence of age, trauma and site of origin of lesions like bone cyst, benign giant cell tumor, osteitis fibrosa and osteoma. Histologic examination also shows that osteoma resembles these fibrocystic diseases. Smith reports 6 cases and analyzes the histologic features of each. He suggests that in addition to trauma possibly some metabolic or nutritional derangement is a causative factor. If the lesion is not definitely neoplastic, perhaps surgical intervention may not be necessary.

Kessel [74] quotes Molan, who in 1938 published a review showing that 458 cases of osteoma had been reported, and in only 11 of these had the growth caused intracranial complications. He cites the following case: A man aged 28 had had a clear colorless fluid discharge from the nose for the past six months. He could stop the discharge by moving his head backward by lying down. Three months before examination he began having headache, vomiting and stiffness of the neck. His condition improved, and he went back to work. The fluid increased in amount, and there were headaches and sciatic pains. Other symptoms were loss of the sense of smell on the right side and slight spasticity of the right leg. A roentgenogram showed an osteoma the size of a walnut in the medial-anterior part of the right orbit, extending into the cranial cavity. The anterior ethmoid cells were cloudy. A great deal of air was seen in the epidural and subdural spaces of the right frontal region and in the third and lateral ventricles. At operation a frontal flap

73. Smith, A. T.: Osseous Lesions of Nose and Sinuses, Arch. Otolaryng. **31**:289 (Feb.) 1940.

74. Kessel, F. K.: Orbito-Ethmoidal Osteomata with Intracranial Complications: Report of a Case, Guy's Hosp. Rep. **89**:837, 1939.

was turned down on the right. Epidural air was encountered. The bone flap was turned down as in an operation for pituitary tumor. The dura was loose and easily retracted from the orbital roof and the osteoma. Later the openings were closed by fascia lata transplants. A broad-based osteoma was removed easily from the roof of the orbit. A roentgenogram made three days later showed the air being absorbed. The patient recovered. The author looked through the literature and found 15 cases of osteoma with pneumatocele on record.

Simpson and Williams [75] report 2 cases and present roentgenograms and diagrams showing a tumor in situ in the operative field. The first case is that of a woman aged 34 who had colds and frequent headaches. Roentgenograms showed localized areas of density in the right frontal sinus. At operation 2 bony tumors, lying in the floor of the frontal and ethmoid sinuses, were exposed. One was the size of a hickory nut, the other the size of a pea. Microscopic examination showed compact bone and small and large haversian spaces. The bone was composed of lacunas with radiating canaliculi. In the lacunas were small bone cells. The surface was covered with fibrous periosteum. The second case was that of a man aged 37 who had headaches and stuffiness of the nose for four years. When he moved his head, he experienced a sensation of something loose in his nose. A polypoid mass in the nose completely obstructed the airway. A roentgenogram revealed cloudiness of the frontal and ethmoid sinuses and of the antrum and a localized area of density in the right frontal sinus. At operation a bony mass was discovered in the posterior medial aspect of the frontal sinus near the septum. It was firmly attached by a bony pedicle. The sinus, which was lined with degenerated mucosa was cleaned out. The tumor was of compact bone.

Rawlins [76] favors the theory advanced by Fetissof as to the origin of osteoma—i.e., that it develops from periosteum split off during the developmental stage of the organism, and that some external irritant may have to be present. He reports the case of a man aged 35 who had painless swelling of the right side of the face and palate ten years. This hard rounded swelling occupied the right canine fossa from the orbital rim down to the alveolar process. Roentgen examination showed an extensive growth. This was removed by the sublabial approach. It was the size of a large walnut and was attached to the alveolus and the anterior antral wall. It was covered with normal mucosa. Histologically the tumor showed a pleomorphic picture: areas of closely packed spicules of cancellous bone with few cells and poorly defined lacunas, separated by fibrous tissue with small cells and much collagen. Some

75. Simpson, W. L., and Williams, R. I.: Osteoma of the Nose and Accessory Sinuses with Report of Cases, Ann. Otol., Rhin. & Laryng. **49**:949 (Dec.) 1940.

76. Rawlins, A. G.: Osteoma of the Maxillary Sinus, Arch. Otolaryng. **32**: 499 (Sept.) 1940.

of this fibrous tissue showed osseous metaplasia but not much osteoblastic activity. The general impression was that the tumor was the result of bony metaplasia from fibrous tissue.

Cyst-Mucocele.—Cora Eliseht [77] contributes a rather extensive article on mucous cysts, which goes into the details of their history, theories of genesis, symptoms, diagnosis and treatment. He seems to lean to the theory that these cysts are inflammatory in origin, arising from dental caries or from dead teeth. An infectious granuloma or local point of irritation about a tooth may stimulate epithelial rests to proliferation, resulting in growth in which the products of exfoliation and degeneration accumulate to form a typical cyst lined with pavement epithelium and containing a fluid with a high percentage of cholesterol. The details of various arguments are given, and the differential diagnosis is elaborated on. The treatment is like that practiced in the United States with a few minor technical differences. The author points out the importance of diagnosing secondary suppuration in the sac and concomitant sinusitis. He emphasizes also the importance of thorough eradication of the cyst with its sac, extraction of the offending tooth and adequate curettage of the alveolar process to prevent recurrence.

Schenck [78] reports a case in which an infected cyst involved four devitalized upper teeth, encroached on the antrum and gave rise to episcleritis and keratitis in a woman of 25, who sought relief for pain in the eye, photophobia and redness. The true condition was diagnosed by roentgen examination, and surgical removal of the cyst was followed by prompt recovery.

Hughes and Dryden [79] report a case in which a dental cyst developed on an inflammatory basis, growing rather rapidly within a year after an infection about a bicuspid tooth. The tumor was diagnosed by roentgen examination and aspiration. The presence of considerable cholesterol (80 mg. of cholesterol per hundred cubic centimeters of fluid) was revealed. The cyst filled the antrum and bulged toward the nasal cavity. The antral walls were thinned by the pressure. The lining of the cyst was thin squamous epithelium, which in places was replaced by granulation tissue. Fragments of bone and mucous glands were also identified. The authors differentiate between secretory, dental and mesothelial cysts according to origins and mention the different types of contents as points in differentiation. McGregor is cited in support of this classification.

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77. Cora Eliseht, F.: Quistes paradentarios ubicados en el seno maxilar, Rev. brasil. de oto-rino-laring. **8**:229 (July-Aug.) 1940.

78. Schenck, C. P.: Infected Radicular Cyst of Antrum as Etiologic Factor in Eye Disease, Texas State J. Med. **35**:567 (Dec.) 1939

79. Hughes, T. E., and Dryden, J. S.: Cysts of the Maxillary Sinus, Virginia M. Monthly **67**:687 (Nov.) 1940.

Kubo [80] again describes postoperative cyst of the cheek appearing years after a radical operation on the antrum. Many cysts of this type have been reported in the Japanese literature and recorded in my reviews for the past five or six years. Kubo states that when he reoperated, he usually found the cyst within the cheek, outside of the antrum, with which it may communicate by means of a contracted fistula through the bony opening into the antrum. From his histologic studies he states definitely that a cyst of this type is not to be confused with a mucocele, which develops primarily in the sinus. Incidentally he states that no cyst of this kind has ever developed after any of his own operations and that most of those reported occurred in cases in which operation had been performed by new assistants. In almost every instance there was a record of severe hemorrhage at the time of operation. [It must be concluded from Kubo's paper that cysts such as these can be avoided by careful hemostasis and proper regard for the delicacy of the soft tissue; in other words, avoid rough handling. S. S.]

Boku [81] conducted a number of experiments on cats, with the idea of discovering the cause of the so-called postoperative cyst of Kubo. 1. All the mucosa of the frontal sinus was removed. 2. All the mucosa was curetted loose but left in the cavity. 3. Part of the sinal mucosa was left in situ undisturbed. He found that cysts developed in the mucosa left behind, portions of which became encysted in the scar. He concludes that cysts such as these can be prevented by complete exenteration of the entire sinal mucosa.

Konishi [82] reports 2 tumors, one of which he calls a mucocele and the other a postoperative Kubo cyst. The first case was that of a man aged 30 who had a radical operation on the antrum two years previously. There were recent pain and swelling in the cheek. A fluctuating mass was felt, which on puncture yielded yellow fluid. At operation the canine fossa was found filled with dense fibrous tissue. The antral mucosa was thin. The counteropening was closed. The lateral wall of the antrum and the lower margins of the orbital bone were diseased. The cyst contained dark brown mucoid secretion, with mucin and cholesterol. The second case was that of a man aged 36 who had a bilateral radical operation on the antrum five years previously. There were swelling and pain in the cheek. Puncture of the swollen cheek yielded thin brown-

80. Kubo, I.: "Postoperative Wangenzyste" (Kubo)- Wangenzyste als eine Spätkomplikation nach Radikaloperation der Kieferhöhleneiterung, Jap. J. M. Sc., XII, Oto-Rhino-Laryng. **3**:43 (Oct.) 1939.

81. Boku, E.: Experimentelle Studien über die Entstehung der postoperativer Wangenzyste nach Kubo, Hukuoka acta med. (Abstr. Sect.) **33**:1 (Jan.) 1940.

82. Konishi, T.: Postoperative Mucocele der Kieferhöhle und postoperative Wangenzyste (Kubo), Oto-rhino-laryng. (Abstr. Sect.) **13**:104 (Feb.) 1940.

ish fluid mucin, cholesterol and diplococci. A cyst was found outside the bone, in the scar tissue of the cheek. The antrum also was filled with a cyst.

The case reported by Bilchick [83] was both interesting and instructive. A man aged 52 complained of lessened vision in the left eye of one month's duration and of dull ache on movement of the eye. Examination showed hyperemia of the left disk, blurring of the margin and engorgement of the veins. An enlarged blindspot progressed in the course of a week to such a point that there was only light perception. Nasal examination did not reveal any abnormality. Roentgenograms showed involvement of the sella turcica on the left side, with a defect which seemed to extend into the sphenoid and posterior ethmoid cells. It was first assumed that a malignant tumor was present, and an attempt was made to obtain a biopsy specimen from the affected region, through the nose. Later the middle turbinate was removed and the sphenoid sinus opened. This was followed immediately by a gush of yellow fluid, leaving a large cystic membrane in the sinus. The anterior wall of the sinus was taken down and the lining removed. On section this was seen to be a fibrous membrane lined with layers of cuboid cells. Some evidence of osteoid activity was seen. The postoperative course was uneventful, and the patient's vision was restored.

Richter [84] cites 4 cases of mucocele of the frontal sinuses. The patients were elderly persons. In each instance the mucocele was characterized by pulsation, which was an indication of absorption of the posterior wall and contact with the dura. In most of the cases there was a history of trauma and long duration. Roentgenograms revealed the extradural location preoperatively. Richter emphasizes the importance of pulsation as an indication of contact with the dura.

De Resende [85] reports 4 cases of mucocele, in 1 of which the antrum was involved. The first case was that of a man aged 72 with a huge swelling above the right eye, which developed in a period of three years. Operation disclosed a large sac containing dark brown fluid, which had eroded the anterior, inferior and posterior walls of the frontal sinus and most of the ethmoid cells. The dura was exposed over a large area. Recovery, with a good cosmetic result, was observed. The second case was that of a woman aged 62 with the complaint of headaches for fifteen years and nasal obstruction on the left. For the past twenty days a fistula had been present at the middle of the left orbital border. Numerous nasal polyps had been removed intranasally some time previously. Pus

83. Bilchick, E. B.: Diseases of the Sphenoid Sinus with Report of a Case of Cyst of the Sphenoid Sinus, Arch. Otolaryng. **32**:1031 (Dec.) 1940.

84. Richter, H.: Die pulsierende Mukokele, Arch. f. Ohrenh. **147**:61, 1940.

85. de Resende, E.: Mucocele fronto-etmoidal, frontal e maxilar, Acta med., Rio de Janeiro **5**:367 (June) 1940.

and polyps were found in the nose. Roentgen examination showed cloudiness of the maxillary and frontal sinuses. Operation disclosed a large frontal sinus with temporal extension. The right frontal sinus contained a mucocele, which was removed. The third case was that of a youth aged 19 who had had swelling of the left side of the face for the past few months. Puncture revealed chocolate-colored viscid fluid. When a Caldwell-Luc operation was performed, it was seen that the anterior wall of the antrum was destroyed by the growth. A membranous sac containing a large amount of chocolate-colored fluid filled the antrum. The ostium was completely blocked. In the fourth case a mucocele of the frontal sinus was encountered in a boy aged 10 years.

Weiss [86] reports 2 cases. The first is that of a woman of 62 who had a swelling over the left eye, which developed slowly for six years, reaching enormous proportions (10 by 8.5 cm.) and projecting 6 cm. from the frontal plane. The eye was markedly displaced and had little vision left. At operation it was found that a tumor mass had eroded the anterior wall of the frontal sinus over an area 6 by 4 cm. and the entire roof of the orbit. The posterior wall of the sinus was not involved. The second case was that of a man of 21 with a history of sinal disease for nine years following an attack of scarlet fever. There was a small elastic mass at the internal angle of the orbit, with slight displacement of the bulb. Operation disclosed a defect in the floor of the frontal sinus. A mucocele occupied the frontal and ethmoid sinuses and had eroded the lacrimal bone and part of the lamina papyracea. The interfrontal septum was absent. A good patent duct was established, as shown by examination a year later. The interesting features in the first case were the presence of several bony septums in the sinus, the long-standing optic atrophy from pressure and the fact that the sac was very thick.

Pimentel [87] saw a mucocele of the frontal sinus eroding into the orbit, causing exophthalmos without the patient's having had any symptoms of inflammation or pain. The onset was gradual. At operation he found a deep backward extension of the sinus and blocking of the duct by polyps.

Miscellaneous Tumors.—Errecart and Tolosa [88] report 4 cases of solitary nasal polyp and present roentgenograms. They discuss the genesis of the growth and produce drawings to show that choanal polyps in the nose are produced by edematous enlargement of the mucosal duplication at the hiatus, coming from the border of the uncinate process,

86. Weiss, J. A.: Mucocele of the Frontal Sinus, Arch. Otolaryng. **32**:1090 (Dec.) 1940.

87. Pimentel, P.: Mucocele frontal e exoftalmia, Rev. méd. brasil. **4**:65 (June) 1939.

88. Errecart, P. L., and Tolosa, E.: Polipo antrocoanal, Rev. Asoc. méd. argent. **54**:118 (Feb. 15-29) 1940.

the maxillary ostium or an adjoining ethmoid cell. They believe that this finding represents a constitutional dyscrasia based on pituitary dysfunction plus local chronic irritation. They also believe that recurrences will follow unless the polyp-bearing mucosa in the middle meatus is removed, which is best done through a transantral approach, since the antrum can be dealt with at the same time.

Terra [89] reports a case of choanoantral polyp in a child of 8 years. Since a roentgenogram showed the antrum dark, he did a Caldwell-Luc operation and found the antral mucosa degenerated and polypoid. The pedicle of a large polyp was located on the posterior wall of the sinus. The polyp extended back to the nasopharynx and down to the palate; it was removed with Brünning's forceps and was the size of a small hen's egg. The operation was done with the patient under anesthesia induced with evipal sodium (the sodium salt of n: methyl-cyclohexenyl-n-methyl barbituric acid) and lasted twenty-five minutes. Recovery was complete in eight days.

Brunner [90] reports a fibromyoma of the ethmoid sinus in a girl aged 12 years. For the past year or more there had been swelling of the right side of the nasal root, causing displacement of the internal canthus outward, widening of the right side of the nasal bridge and displacement of the bulb. There was also a fungating mass about the middle turbinate. The taking of a specimen for biopsy was accompanied with moderate bleeding. Roentgenograms revealed cloudiness of the frontal and ethmoid sinuses and antrum. An external operation was performed, and a large mass involving the entire ethmoid capsule was removed. The lamina papyracea was destroyed. The mass extended almost to the apex of the orbit. Recovery followed with the formation of a small external fistula. A detailed histologic examination revealed fibrous tissue, atypical striped and unstriped muscle fibers and hyaline and amyloid deposits. The muscle tissue was assumed to have its origin in mesoderm of the second branchial arch where the platysma and orbital muscles take their origin. Misplaced cells may be carried upward into the ethmoid capsule to form a nidus for the development of a tumor.

Monteiro's [91] case was that of a 24 year old patient whose symptoms were nasal obstruction and loss of the sense of smell of several years' duration. A tumor measuring about 6 by 2.5 cm. arose in the middle meatus and gravitated toward the choana. It was removed through the nasopharynx by means of a snare after the palate was elevated. Monteiro

89. Terra, M.: Polipo antro-coanal na creança, Rev. brasil. de oto-rino-laring. **8**:159 (May-June) 1940.

90. Brunner, H.: Fibromyoma of the Ethmoid, J. Laryng. & Otol. **55**:23 (Jan.) 1940.

91. Monteiro, A.: Fibroma do etmoide, Acta med., Rio de Janeiro **6**:33 (July) 1940.

in a review of the literature finds that cases such as this are rare. The only possible confusion in diagnosis might arise in cases in which the tumor becomes so large that its source cannot be definitely established. In these cases the growth might be mistaken for a true nasopharyngeal fibroma.

Balzer [92] presents 2 cases of true fibroma of the nasal sinuses, with histologic studies. The presence of calcareous deposits often makes this tumor appear like osteoma, for which it may be mistaken. Balzer claims that this tumor is rare and cites several authors in support of his contention. The first case was that of a girl aged 16 with a history of slight exophthalmos in the right eye for the past eight years. For the past two years there had been more rapid growth of the tumor, with increasing exophthalmos, as well as external displacement. There were also marked nasal block, headache and loss of weight. The right nostril was blocked, and the septum pushed to the left. A hard mass was felt with a probe. Roentgen examination showed a dark shadow in the right ethmoid sinus and antrum, with areas of lighter density. The floor of the orbit was pushed up and outward. The sharp outline of the tumor suggested osteoma. A Kuhnt operation was performed. The tumor removed was irregular and hard, and when cut it showed numerous kernels of grayish white masses, which were hard. The base of the tumor was about 1.5 cm. in diameter and was attached to the median ethmoid area. Histologic examination revealed a covering of delicate squamous cell epithelium and dense subepithelial fibrous layers, rich in vessels. There were also masses of calcareous deposits in a netlike arrangement. Small spaces were found lined with a layer of endothelium. Areas of fibrous tissue showed hyalinization, evidence of beginning calcareous degeneration. Balzer believes with Chiari and others that tumors such as this originate from the periosteum. In a second case a tumor of similar nature was found in the frontal sinus, with secondary infection of the sinus.

Takahashi [93] reports the case of a child of 10 years who for two and a half years had nasal obstruction and swelling of the right cheek and palate. The septum and left nasal passage showed involvement by a tumor. There was no pain or tenderness, and no glands were palpable. Roentgenograms did not show any areas of density. The tumor filled the right antrum, extended into the ethmoid labyrinth and pressed the septum and the palate out of shape. The tumor substance (which actually invaded the last-named structures) consisted of numerous round, small sandlike and large bony islands. After histologic examination the tumor

92. Balzer, R.: Zwei Fälle von echtem Fibrom der Nasen-Nebenhöhlen, Ztschr. f. Hals- Nasen- u. Ohrenh. **45**:307, 1940.

93. Takahashi, T.: Ein Fall von multiplem Osteofibromyxom der Kieferhöhle, Oto-rhino-laryng. (Abstr. Sect.) **13**:24 (May) 1940.

was diagnosed as a multiple osteofibromyxoma. It was totally extirpated through a Denker approach.

Hickey [94] reports a chondroma of the ethmoid sinus in a woman of 56, who was observed for a period of eighteen years. The chondroma was seen originally in the infraorbital region, with extension into the nose. An intranasal operation gave the patient relief until 1927. During the next nine or ten years she suffered several attacks of orbital abscess, acute dacryocystitis and local recurrences within the nose. In 1933 a Caldwell-Luc operation was done, and several years later the patient had to submit to a modified Lynch operation. The floor of the frontal sinus and the external wall of the ethmoid sinus were found to be necrotic. A histologic examination revealed a typical cartilaginous tumor surrounded by inflammatory tissue.

Keegan and Baker [95] report a giant cell tumor of the frontal sinus in a boy of 13. About two years before examination a protruding mass was noticed on the forehead. Gradually it became larger. It was firm, painless and not tender. It encroached on the right eye, displacing the bulb and restricting ocular movements. There was no history of trauma. The mass was 5 cm. in diameter and was elevated 1 cm. above the level of the bone. It was just above the supraorbital margin and the external angular process of the frontal bone. It extended laterally across the temporal ridge into the temporal fossa. There was slight exophthalmos. Roentgenograms showed an extensive area of decalcification of bone and a thin septum separating the tumor from a small frontal sinus. Before operation the growth was diagnosed as a mucocele. At operation the anterior table of the frontal bone was found found to be very thin. Rather firm gray tumor tissue filled the defect and was diagnosed immediately as either xanthoma or giant cell tumor. Considerable hemorrhage attended its removal. The inner table was thin and eroded in places. On account of the extensive hemorrhage, necessitating the application of an electrocautery, the tumor was not completely removed. Roentgen therapy was given—1,035 r in divided doses. The patient remained well for two years after the operation. On section the tumor showed great numbers of multinucleate giant cells, as many as twenty-five nuclei usually tending to group near the center. These cells were more numerous about the periphery of the mass. Keegan and Baker have no theory as to the genesis of the tumor but quote Geschickter and Copeland as attributing it to a resorptive process in cartilaginous bone.

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94. Hickey, H. L.: Chondroma of the Ethmoid, Arch. Otolaryng. **31**:645 (April) 1940.

95. Keegan, J., and Baker, C. P.: Giant Cell Tumor of the Frontal Bone, J. Lab. & Clin. Med. **26**:319 (Nov.) 1940.

Monteiro [96] describes a case in which a solitary myeloma of the nasopharynx invaded the sinuses of the affected side. The soft friable, easily bleeding mass filled the nasal cavity, pushing the septum to the opposite side. The choanae were completely blocked, and the maxillary, ethmoid and frontal sinuses were invaded to such a degree that an external bulge was seen on the face. Roentgen examination showed erosion of considerable bone, including the anterior wall of the frontal sinus and the frontal process of the superior maxilla. A radical operation was done with the region under local anesthesia. The Moure exposure was used. Histologic examination showed the growth to be myeloma. After recurrence, roentgen therapy, the administration of vitamin C and blood transfusions were instituted. When the patient was seen eighteen months later, he was free from disease. A detailed histologic examination showed neoplastic proliferation consisting mainly of cellular elements, large round cells with large nuclei and nucleoli. Cells having the characteristics of myeloblasts and numerous mitotic figures were seen. In other areas the structure was more of the plasmocytic variety. In some areas there were numerous new-formed capillaries, and in other areas, signs of local necrosis. The cells, mostly lymphocytes and plasmocytes, were in groups or in nodular form, surrounded by connective tissue septums. [Monteiro cites numerous cases from the literature but does not mention Pollock's case. S. S.]

Dantas [97] reports an unusual peridental teratoma in a child of 7 years. After the appearance of the median superior incisors, an elevation of the surrounding alveolar area was noticed. Later this could be seen externally at the angle of the nose and face. The mass felt elastic to the touch, Dantas says, "like celluloid." When the growth was removed, one incisor was included with it. The mass had eroded into and occupied a large portion of the antrum. Histologically it was composed of calcified osseous areas, muscular fibers and connective tissue and had undergone some cystic degeneration. Clinically the growth appeared to be a peridental cyst, but histologic examination proved it to be a teratomatous neoplasm.

(To Be Concluded)

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96. Monteiro, A.: Mieloma solitario do cavum com invasio dos seios da face, Hospital, Rio de Janeiro **17**:265 (Feb.) 1940.

97. Dantas, J. E. C.: Cisto pericorono-dentario teratomatoso desenvolvido no antro de Highmore, Rev. brasil. de oto-rino-laring. **7**:371 (Sept.-Oct.) 1939.

Abstracts from Current Literature

Ear

TWO YEARS' EXPERIENCE WITH THE AUDIOMETER IN THE SPRINGFIELD, ILL., SCHOOLS. G. KOEHLER, Illinois M. J. **76**:555 (Dec.) 1939.

During the past two years 6,570 children in the schools of Springfield, Ill., were tested with the 4 B audiometer. Three hundred and four, or 4.6 per cent, had defective hearing as measured by the standard used. Of 37,977 school children in Illinois whose hearing was recently tested, 13.1 per cent were found to have defective hearing as measured by the standard used. Of 1,366 students tested in the Springfield high school, 6.2 per cent were found to have defective hearing. The results of retests made after one year tend to show that a certain per cent of the hearing deficiencies are evanescent and are recovered from spontaneously.

BARBOUR, Peoria, Ill. [AM. J. DIS. CHILD.]

PREVENTION OF PURULENT OTITIS MEDIA IN INFANTS. HARRY BAKWIN and HAROLD JACOBZINER, J. Pediat. **14**:730 (June) 1939.

From 1934 to 1938 the management of otitis media in infants under 2 years of age in the children's medical service of Bellevue Hospital was carefully supervised, and the following indications for myringotomy were set up: complete obliteration of the landmarks, especially of the short process, and a bulging gray drum, together with fever, restlessness and disturbed sleep. In the presence of intense pain, great restlessness and loss of sleep these criteria were modified. Frequent examinations were discouraged, because of the danger of transmission of additional infection from the examiner to the patient and because it was felt that manipulation in the canal, by traumatizing an already inflamed drum, might aggravate the condition. All infants were examined while they were lying on the abdomen with the head turned away from the examiner. Myringotomy was performed only with the approval of an attending physician.

The incidence of purulent otitis media from 1934 to 1938 was compared with that during a control period (1931 to 1933). During the control period the incidence was 23.3 per cent of all patients admitted, whereas from 1934 to 1938 it was reduced to 8.9 per cent. During the control period 1.8 times as many infants had bilateral purulent otitis media as had the unilateral condition; in the period of supervision the patients with the unilateral condition outnumbered those with the bilateral. The incidence of purulent otitis media among the infants with diarrheal diseases and with pneumonia decreased to less than one third during the period of supervision. The number of infants operated on for disease of the mastoid was reduced from 1.38 per cent of all ward patients admitted during the control period to 0.56 per cent for the period of supervised management.

RAUH, Cincinnati. [AM. J. DIS. CHILD.]

PEDIATRIC TREATMENT OF THE ACUTE EAR. NORMAN WARD CLEIN, Northwest Med. **39**:135 (April) 1940.

Clein points out the difference between pediatric management and otologic treatment of otitis. The objectives of treatment in cases of infection of the upper respiratory tract are: (1) prevention of the spread of the infection to the ears, (2) conservative medication of the inflamed ear, (3) paracentesis if necessary and (4) careful observation and patience in treating the complications that may arise from a draining ear.

Sulfanilamide treatment decreases the duration of the discharge by one half to one fourth, as compared with the customary treatment.

Sulfapyridine (2-[paraaminobenzenesulfonamids]-pyridine) seems to be more effective in some of the cases in which a ready response to sulfanilamide is not obtained.

The author states that since sulfanilamide has been available he has not had a single case in his private practice in which he felt it necessary to institute surgical treatment of the mastoid. ROBERTSON, Omaha. [AM. J. DIS. CHILD.]

THE SCHILLING HAEMOGRAM IN OTITIS MEDIA AND MASTOIDITIS OF INFANCY. EDWARD C. ROSS COUPER, Arch. Dis. Childhood **15**:27 (March) 1940.

Ordinary white blood cell counts and differential counts are of no value in prognosis or differential diagnosis of otitis media and mastoiditis in infants. Analysis of the polymorphonuclear cells on the lines suggested by Schilling and others is applicable to infants under the age of 1 year. The use of some index of "left shift," such as the multiple index employed in the series reported, is possibly of diagnostic value and certainly of prognostic value. Single counts are not nearly so useful as a series of estimations. The height of the multiple index is of no value as an indication of the time to operate, for each infant's response to the infection varies, and such counts must always be considered in conjunction with the clinical condition. An infant's weight has long been regarded as a good indication of its progress, and the manner in which the multiple index rises with loss of weight and falls with gain in weight is striking. As the multiple index and kindred indexes are established as reliable indexes of infection somewhere in the body and as the multiple index in the series reported fell and rose according to the success or nonsuccess of treatment directed against the aural infection, it seems reasonable to conclude that the aural infection was the immediate cause of the infant's illness. KELLY, Milwaukee. [AM. J. DIS. CHILD.]

DEFECTIVE HEARING AND NUTRITION IN CHILDREN. PHYLLIS M. TOOKEY KERRIDGE, GWENDOLINE BRIGGS, D. P. JOYCE and JANET HILL, Lancet **2**:781 (Oct. 7) 1939.

Severe deafness is not common among children. In England about 0.1 per cent of children attending public elementary schools are deaf enough to require special educational provision.

In a survey of the London children in the schools for the deaf or partially deaf it was found that about half of them were born deaf and that about a third were deaf as a result of disease of the middle ear.

All the children tested in this study were between 7 and 14 years of age; one group was chosen from good environments and the other from poor environments. It was found that disease of the middle ear is about four times as common under poor social conditions as it is under good; in the poorest places it may be nearly ten times as common.

As a result of their study the authors feel that probably the most important work on the prevention of deafness will be done by those who are striving to improve the social conditions and in particular the nutrition of the nation.

LANGMANN, New York. [AM. J. DIS. CHILD.]

ETIOLOGY AND CLINICAL PICTURE OF ACQUIRED DEAFNESS. J. KOCH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:267, 1939.

Koch reports the cases of 33 patients with acquired deafness, 7 of whom had suppuration of the middle ear and associated labyrinthitis. Of the remaining 26, 18 had meningitis of varied cause (cerebrospinal infection, trauma, pneumonia, measles, scarlet fever, grip and typhoid). Six had deafness of a toxic nature. The deafness of 1 was due to syphilis, and 1 had myelogenous leukemia. In 19 the deafness showed a direct relationship to an acute infectious disease. In consequence of this analysis, the author believes that the most common cause of acquired deafness is meningitis, either latent or frank.

As a result of his studies, he further believes that a bilateral loss of hearing with a normal vestibular apparatus (as found in hereditary deafness) is never observed in persons with acquired deafness. On the contrary, an unequal impairment of hearing is often associated with an impairment of vestibular function. This impairment of vestibular function is disproportionate for the two ears, and usually disproportionate to the amount of impairment of hearing, and it nearly always occurs as a late symptom.

PERSKY, Philadelphia.

TRAUMATIC RETROLABYRINTHINE SYMPTOM COMPLEX. RUF, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:291, 1939.

The symptoms following injuries to the skull include, in addition to disturbances of hearing and equilibrium, disturbances of other cranial nerves. These disturbances are central rather than peripheral. While certain nerves may be involved and the site of lesion easily localized, occasionally the sensory division of the trigeminal nerve is the only one affected. When this occurs, it may be difficult to determine the exact nature of the lesion.

The author employs a method of measuring the sensitivity of this nerve. He has devised a barbed stylet, which is graduated in grams of pressure. He impinges the point of the stylet on the mucous membrane of the nose. A normal patient will complain of pain under a pressure of 1 Gm. However, when there is anesthesia of the mucous membrane due to a lesion involving the sensory division of the trigeminal nerve, the pressure may be increased to 10 Gm. without any response from the patient. Should this occur, the author believes the fact is of diagnostic value, claiming that the lesion is usually in the retrolabyrinthine space, probably in the brain stem. Since these lesions may be of only a minimal degree and all other localizing signs and symptoms may be absent, he believes that by this test a lesion can be readily localized in this region.

PERSKY, Philadelphia.

CLINICAL CONSIDERATIONS OF THE ROTATIONAL TEST. MITTERMAYER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:305, 1939.

In discussing the value of the rotational test, the author first tries to evaluate the differences in the modes of performing the test. He compares the results obtained by a slow and by a sudden stopping of the Bárány chair. He reaches the conclusion that the responses after a slow stop are slower in onset, somewhat weaker in appearance and shorter in duration. However, this observation is of no real significance and should take a role secondary to the direction of the nystagmus. On the other hand, his results show that postrotational vertigo is definitely influenced by the method of stopping the chair.

He considers nystagmus as being composed of two phases: (1) a rapid movement and (2) a slower movement. By means of graphs he has shown that when the chair is stopped slowly the peak of the first phase is low and the second phase appears rather quickly. On the other hand, when the chair is stopped suddenly the first phase is definitely higher and more prolonged (greater number of oscillations) and when the second phase occurs it is definitely prolonged. The author considers that the ratio of the strength of the second phase to that of the first is an important index as to whether the lesion is peripherally or centrally located, and he postulates that when there is an equal variation from normal in the two phases the lesion is peripheral, but when there is a disproportion the lesion is central.

The magnitude of the first phase depends not on the strength of the physical stimulus but on the responsiveness of the peripheral organ. The degree of this responsiveness is expressed by the two components. He considers the second phase as evidence of a response of the central nervous system, which is more or less influenced by the size or the degree of the impulse from the periphery.

He considers this quantitative method of value in determining the location of a lesion, either peripheral or central, and he presents a number of cases wherein he has been able to establish a definite localization of the lesion.

PERSKY, Philadelphia.

A New Method of Treatment of Othematoma. A. Herrmann, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:363, 1939.

Herrmann recommends a crescentic incision of the posterior surface of the auricle. He excises a small fragment of cartilage and evacuates the blood clot thoroughly. He cleanses the wound and closes it with sutures.

He prefers this technic in that it is aseptic and the wound can be closed immediately. This procedure also permits a more rapid resorption of the blood clot, and, because of the posterior incision, the scar will contract posteriorly and so favor a better cosmetic result.

Persky, Philadelphia.

Pharynx

The Roentgen Diagnosis and the Therapy of Retropharyngeal Adenitis. R. Rhett Rathbone, Am. J. Roentgenol. **43**:25 (Jan.) 1940.

A series of lateral roentgenograms taken during inspiration in 80 healthy children were examined, and it was found that the normal retropharyngeal space in infants and children does not measure over 11 mm. by ordinary roentgen distances. Retropharyngeal adenitis was diagnosed in 20 instances on the basis of enlargement of the retropharyngeal space. This condition was usually due to some focal infection in the upper respiratory tract, often in the nasal accessory sinuses. Of the 20 patients, 17 were treated with roentgen therapy, with complete disappearance of the retropharyngeal swelling in all cases. Both the area of swelling and the focus of infection should be treated by roentgen therapy.

Squire, Chicago. [Am. J. Dis. Child.]

Disorders of the Mouth of the Oesophagus in the Syndrome of Plummer-Vinson (Dysphagia with Anaemia). P. G. Gerlings, J. Laryng. & Otol. **55**:143 (March) 1940.

The association of hypochromic anemia and dysphagia (Plummer-Vinson syndrome) has for a long time interested many laryngologists.

Most of the patients are middle-aged women, whose complaint is localized at the level of the larynx. On examination the most typical symptoms are a small mouth with fissures in both corners, edentulousness and a pale buccal mucosa. The mucosa of the tongue is glossy and sometimes has areas of leukoplakia. The mucosa of the pharynx is dry and often atrophic. These symptoms are accompanied with a hypochromic anemia called "simple achlorhydric anemia." Koilonychia (spoon nail) is sometimes present. Oral administration of iron and, if necessary, of hydrochloric acid gives surprising results.

Roentgen examination shows typically stagnation in the valleculae epiglotticae and in the piriform sinuses, stagnation just above the mouth of the esophagus, spastic stricture below the mouth of the esophagus or cicatricial stenosis of the esophagus.

Esophageal examination shows spasms, webs, areas of cicatricial stenosis, areas of granulation tissue and sometimes spots of leukoplakia.

It is important to perform esophagoscopic examinations in cases of this condition, in order to determine the possibility of carcinoma in association with the other lesions.

LeJeune and Bayon, New Orleans.

Bacteremia Following Tonsillectomy. S. D. Elliott, Lancet **2**:589 (Sept. 9) 1939.

Transient bacteremia was observed in 38 of 100 patients within a few minutes of tonsillectomy. The organisms isolated included Streptococcus pyogenes, Streptococcus viridans, Streptococcus pneumoniae, Haemophilus influenzae, Staphylococcus and Corynebacterium. Serologic matches for Str. pyogenes recovered from the blood were isolated from the corresponding tonsils after their removal. In 64 of the 100 cases studied and in 18 additional cases blood for culture was also taken

immediately before operation. Of the 82 cases, positive blood cultures were obtained before operation in 7. In 3 of these 7 the postoperative blood culture was positive.

The possible bearing of postoperative bacteremia on the causation of subacute infective endocarditis is discussed.

LANGMANN, New York. [AM. J. DIS. CHILD.]

Larynx

CHRONIC STENOSIS OF THE LARYNX WITH SPECIAL CONSIDERATION OF SKIN GRAFTING. FREDERICK A. FIGI, Ann. Otol., Rhin. & Laryng. **49**:394 (June) 1940.

The causes and treatment of stenosis of the larynx are discussed. Treatment for chronic stenosis of the larynx is not an emergency measure and should be delayed until the patient is in the best possible general condition and any local inflammatory reaction has entirely subsided. The type of treatment depends largely on the character and extent of the stenosis rather than on the cause. At the Mayo Clinic, bougienage has been the least effective treatment. Elastic pressure applied by means of soft rubber dilators has been the most satisfactory. In chronic cicatricial stenosis the most effective method was laryngofissure, with excision of the scarring and of any thickened or distorted cartilage, followed by skin grafting. The grafts are placed about a sponge rubber core before being inserted. The technic of the operation is given.

M. V. MILLER, Philadelphia.

TREATMENT OF LARYNGEAL PAPILLOMA IN CHILDREN WITH ESTROGENIC HORMONE. EDWIN N. BROYLES, Bull. Johns Hopkins Hosp. **66**:319 (May) 1940.

Since papillomas tend to stop growing at or near puberty and since estrogens are known to have the power to change infantile epithelium to epithelium of the adult type, it was thought that local applications of an estrogen to the larynx of children suffering from papilloma might be beneficial.

Five patients were given weekly applications of amniotin in oil. It was estimated that 0.1 cc. (1,000 international units) of the substance was applied to the larynx at each treatment. Broyles reports a definitely beneficial effect and thinks that the results of this simple form of treatment are more satisfactory than those obtained in the past.

LYTTLE, New York. [AM. J. DIS. CHILD.]

PARALYTIC SYNDROME OF THE RIGHT RECURRENT LARYNGEAL NERVE FROM A CERVICAL SCLEROGUMMATOUS PROCESS. G. SAIBENE, Arch. ital. di otol. **52**:475 (Sept.) 1940.

Saibene's report concerns a man aged 46 years who had acquired syphilis twenty-five years previously and who had not been adequately treated. The patient was admitted to the hospital with hepatic cirrhosis and died within a few weeks. Two years before admission, disturbance of the laryngeal function from a hemiparalysis of the larynx on the right had appeared and persisted.

Autopsy revealed, among other things, two hard nodules, the size of large peas, lying between the trachea and the esophagus on the right side, 1.5 cm. below the inferior margin of the cricoid cartilage; the mass of inflammatory infiltration enveloped the recurrent laryngeal nerve. Histologic study of the tissue proved its syphilitic nature.

DENNIS, San Diego, Calif.

A CASE OF CONGENITAL DEFORMITY OF THE LARYNX WITH A FATAL OUTCOME. J. STRÖM and L. TROELL, Acta pædiat. **25**:266, 1939.

The report describes the case of an 11 month old mongoloid mentally defective infant whose early symptoms of congenital laryngeal stridor became progressively worse, were punctuated by episodes of cyanosis and finally terminated in death.

Autopsy revealed a rare anomaly: There was a complete defect of the midportion of the cricoid cartilage, through which an anomalous adhesion between the larynx and the esophagus had been effected. The larynx was unduly soft and mobile. Two similar cases were found in the literature. The embryologic features of the malformation are discussed. In the light of the associated physical anomalies the mental defect was interpreted to be developmental.

McCune, New York. [Am. J. Dis. Child.]

Nose

Diagnostic Pitfalls of the Nasopharynx. Frederick T. Hill, J. A. M. A. **115**:662 (Aug. 31) 1940.

Hill clearly explains the importance of a careful inspection of the nasopharynx and states that the main pitfalls in the diagnosis of nasopharyngeal disease may be: (1) not looking, (2) not seeing, (3) not knowing what to look for and (4) not interpreting correctly what is seen.

He comments on the use of the postnasal mirror and its limitations and on the value of the nasopharyngoscope but states that he places the greatest reliance on direct inspection by elevation of the soft palate with a retractor.

He outlines the numerous pathologic processes that should be searched for in the nasopharynx, especially the inflammatory and exudative processes, hypertrophies, hyperplasias, polypi, nasopharyngeal fibromas, embryologic growths and tumors. He quotes a number of authorities, besides citing several cases, each with different findings, with the ultimate conclusion that he has found direct inspection of the nasopharynx by elevating the soft palate with a retractor the most accurate and helpful method.

Gordon, Philadelphia.

Correlation of Chronic Infection of the Upper and Lower Respiratory Tracts. Ferris Smith, Pennsylvania M. J. **43**:1389 (July) 1940.

Smith presents for consideration and evaluation the following factors established by the observations, researches and statistical compilations of investigators during the past twenty-five years, which tend to render an entity the correlation of chronic infection of the upper and lower respiratory tracts.

1. *The Prenatal and Developmental Factor.*—The important role played by congenital anomalies, prenatal lack of development and predisposition, constitutional tendencies, inhibited development, local anomalies and situs inversus in the causation of respiratory diseases indicates an interrelationship of the divisions of the respiratory tract.

2. *The Anatomic and Physiologic Factor.*—The anatomic construction and physiologic functioning of the three divisions of the respiratory tract tend to show the interrelationship of the divisions.

3. *Pathways and Mode of Infection.*—The lymphatic drainage of the sinuses demonstrated by investigators (Larsell, Fenton and others) produces pulmonary end results.

4. *The Prevalence and Behavior of Respiratory Disease in Children.*—The fact that the lower respiratory tract is not fully developed until the age of 7 has led investigators to realize its predisposition to infection and definitely to prove the high incidence of infection of the upper respiratory tract in infants and children with pulmonary disease.

5. *Sequence of Respiratory Infection.*—The fact that an acute infection manifests itself in one of the divisions of the respiratory tract does not exclude its preexistence in another division. Its localization may be due to anomalies, lack of resistance, absence of a defensive mechanism or other causes.

6. *Frequency of Association.*—Universally renowned observers have reported the frequency of association of chronic sinus disease, bronchiectasis, otitis media and similar infections.

7. *Allergy, the Endocrine System and Diet.*—(*a*) The picture of an intranasal allergic condition may also be seen in the lower divisions of the respiratory tract, where a soil suitable for infection has been prepared and the mechanical blockage of ventilation and drainage hastens the development. The allergic condition is probably due to a physiologic process resulting from a disturbance in fluid concentration.

(*b*) Pathologic conditions from glandular dysfunction play an important part in the causation of respiratory disease. Hypometabolism, adrenal dysfunction and thyropituitary defects influence the development of the respiratory tract; gonadotropic hormones from the pituitary gland specifically affect the respiratory mucosa; estrogen and androgen produce hyperemia of the turbinates, and changes in the male voice and epistaxis at puberty. Acute or chronic tonsillitis causes thyroid dysfunction (Billings).

(*c*) Deficiency of vitamin A or a diet high in vitamins and low in protein causes sinusitis and otitis media in monkeys (Dean). Chronic sinusitis and pulmonary fibrosis associated with hypoglycemia and ketosis (C. Sippe) often have an exudative diathesis as a basis, i. e., inability to retain water in the tissues, due to insufficient dextrose. An excess of fats or of salt results in marked catarrh.

8. *The Mechanical Factor.*—Blockage (anatomic and physiologic), abnormalities and obstructive deformities in the nose and nasopharynx, investigation demonstrates, operate in the lower respiratory tract, producing weak respiratory musculature and feeble cough effort in young children, with retention of tenacious secretions.

BRIGLIA, Philadelphia.

MAXILLARY SINUSITIS. FELIPE CORA ELISEHT, Rev. argent. de oto-rino-laring. **9**:25 (Jan.-Feb.) 1940.

Eliseht first discusses the anatomic relationship of the maxillary sinuses, stressing the neighboring structures, such as the facial bones, the orbit, the other sinuses, the nerves, the blood vessels and the teeth. As a frequent direct cause of maxillary sinusitis he emphasizes the importance of a latent or frank pathologic condition in and about the roots of the teeth and their proximity to the floor of the sinuses.

From a clinicopathologic standpoint, he classifies maxillary sinusitis as (1) acute (catarrhal, purulent or necrosing) and (2) chronic, which may be either simple latent, hypertrophic, serous, catarrhal or purulent with modifications. The author discusses the pathologic aspects of each group in detail, stressing the mode of progress from one stage to the next. He emphasizes the intimate relationship of the symptoms with each pathologic state.

After reporting a number of cases, with a detailed description of the pathologic findings from the material removed from the sinuses, Eliseht concludes that a thorough knowledge of the pathologic anatomy of the sinuses is extremely important, so that proper treatment can be instituted. He believes that his classification helps establish a correct diagnosis and that this, in turn, will minimize a great many errors in treatment.

PERSKY, Philadelphia.

HYPERERGIC REACTIONS OF THE NASAL AND PARANASAL MUCOUS MEMBRANE. EMILIO RICCITELLI, Rev. argent. de oto-rinolaring. **9**:139 (May-June) 1940.

Riccitelli gives an extensive and thorough discussion of many interesting factors which not only are associated with allergy but also influence it.

After evaluating all of these, he expresses the belief that allergy is principally a result of hyperexcitability due to alterations in the autonomic nervous system. He is also impressed by the climatic and other physical factors that play a role in allergy. He believes that allergy develops primarily in the spring and that

cosmic and meteorologic influences play a particularly important role in its production. Temperature changes and both the luminous and the caloric radiations of the sun are important. The electrical state of the atmosphere produces an important effect on the nervous phenomena.

He concludes by stating that careful investigation of all the factors which he has outlined in his survey should be made, so that proper treatment can be instituted.

PERSKY, Philadelphia.

Miscellaneous

GRANULOCYTOPENIA IN SULFAPYRIDINE THERAPY. VERA B. DOLGOPOL and HAROLD M. HOBART, J. A. M. A. **113**:1012 (Sept. 9) 1939.

In reviewing the literature, Dolgopol and Hobart found 3 cases of granulocytopenia and several of leukopenia after sulfapyridine therapy. All the patients were infants who had been treated for pertussis and pneumonia. In the 1 fatal case there were complete disappearance of granulocytes and an aplastic marrow.

Dolgopol and Hobart advise biweekly blood counts during sulfapyridine therapy and for two weeks after withdrawal of the drug. The granulocytopenia and leukopenia in these cases always occurred when large doses of the drug were used over long periods.

HEERSMA, Kalamazoo, Mich. [AM. J. DIS. CHILD.]

THE MANAGEMENT OF ACUTE CRANIOCEREBRAL INJURIES. BERNARD S. BRODY, J. A. M. A. **115**:977 (Sept. 21) 1940.

Brody discusses the management of injuries to the head, emphasizing (1) the prevention of infection, (2) the treatment of increased intracranial pressure and (3) the management of depressed fractures.

When fracture has taken place through the mastoid process or paranasal sinuses, cerebrospinal otorrhea or rhinorrhea may result. Leakage of spinal fluid from the ear or nose usually stops spontaneously. Sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) in the proper doses should be given early, intranasal therapy should be avoided and the patient should be cautioned against sneezing or otherwise raising the pressure of the spinal fluid. Intra-aural examinations should be avoided if there is bleeding or leakage of spinal fluid from the ear. Instead, the ear should be covered with a sterile dressing and kept dependent, to encourage external drainage. The foot of the bed should be elevated about 12 inches (30.5 cm.), for this measure has been found of value in decreasing the incidence of aspiration pneumonia in patients who are in deep coma.

The management of the other conditions cited belongs to the fields of neurology and neurosurgery, and the principles outlined therein are outside the field of otolaryngology. Roentgenograms, charts and visual fields accompany this article, enhancing the illustrative case reports.

GORDON, Philadelphia.

THE PROBLEM OF LIPID PNEUMONIA. PAUL R. CANNON, J. A. M. A. **115**:2176 (Dec. 21) 1940.

At this year's meeting of the Council on Pharmacy and Chemistry of the American Medical Association, the referee for preparations used nasally called attention to the increasing number of reports of lipid pneumonia caused by the use of oily preparations in the nose. Dr. Cannon was asked to prepare a paper on this subject, the Council adopting the review for publication. The article is the subject of this abstract.

A member of the department of pathology of the University of Chicago, Dr. Cannon has done a great deal of work on lipid pneumonia but in preparing this paper has drawn freely from the literature of all the specialties. His review is thorough and complete.

Though a number of terms have been ascribed to the type of pneumonia due to the presence of oil in the lungs, the best is "lipid pneumonia," as suggested by Graef.

The fact that unsuspected lipid pneumonia continues to be found at many necropsies despite the clarification of this subject, or certain phases of it, during past years suggests that the disease is still so unfamiliar to many, even physicians, that a thorough review is in order.

The pneumonia varies in severity, depending on the kinds and amounts of lipids aspirated. Some oils are markedly irritating and cause acute edema and alveolitis with accompanying symptoms of cough and increased respiratory rate. Massive aspirations of oil have led to attacks of choking, cyanosis and suffocation, caused by the accumulation of large amounts of oil in the alveolar spaces. Particularly serious is the septic pneumonia following aspiration of irritating oil and pathogenic micro-organisms. Occasionally the effects on the pulmonary tissue are minimal, with slight symptoms, as when small amounts of liquid petrolatum enter the lungs. But this becomes serious in due time, because of the prolonged irritating effects of the accumulated oil in the lungs and the insidious development of permanent deep-seated changes.

The most important lesson to be learned from the study of lipid pneumonia is the ease with which light oils and fluids can pass from the upper respiratory passages to the lungs.

The lipids may be introduced either by mouth or into the upper respiratory passages. The first group includes cod liver oil, halibut oil, castor oil, liquid petrolatum or milk fat; the second group comprises the various medicated oils that are used as intranasal sprays or nose drops.

Persons of any age may be affected. The former view that lipid pneumonia can occur only in debilitated persons, especially those with defects of deglutition, is no longer held. There is sufficient evidence at hand to prove that severe types of lipid pneumonia may develop in healthy persons who use medicated liquid petrolatum intranasally in large quantities over long periods.

Babies, debilitated adults who are recumbent, patients having cleft palate, convulsions, spasms or frequent attacks of gagging and vomiting, those with cerebral birth injuries and congenital neurologic disorders or those suffering with brain tumors or local inflammations or neoplasms of the mouth, throat, larynx or esophagus are often likely to aspirate these lipids.

Extensive advertising campaigns by makers of various nose drops and sprays have done much to encourage patients to use such medicaments at home. Self medication has been furthered. Patients have been persuaded to instil a prescribed amount of "drops" or "spray" for "that stuffy head" and other symptoms of cold. Many have been relieved by these methods and have continued to use them. Others, who have not been relieved, have noted no harm from the practice. Patients are never warned by the advertisers of the harmful effects of continued use of the nasal oils.

Physicians have done much to guide their patients, but the latter have acquired the "habit" of following advice given by radio and numerous other sources of advertising, to the end that injudicious and misguided methods of home medication have caused cumulative pulmonary damage.

Liquid petrolatum is used as the solvent for various medicaments because it is said to be bland and nonirritating, not to become rancid, to spread easily and to prevent drying and crusting of the respiratory surfaces. Though all of these things are to a certain extent true, it must never be forgotten that this oil, being light, does not elicit the cough reflex and under certain conditions can enter the lungs by aspiration, after which it will gravitate slowly into the alveolar spaces.

Cases have been reported in which this was proved to be true; e. g., when the oil was instilled while the patient was in a recumbent posture and in infants.

Once liquid petrolatum has entered the alveoli, it becomes far from bland. Though less damaging than any of the other oils, including those of the animal and vegetable groups, it produces varying lesions, ranging from simple congestion to edema and necrosis. Authorities have again and again pointed out that several of the vegetable oils, notably croton oil, castor oil and peanut oil, are exceedingly active tissue irritants and that chaulmoogra oil is markedly toxic to pulmonary tissues.

Menthol often acts as a mild anesthetic, thereby favoring the passage of the oil into the lungs.

The most unfortunate aspect of the entire problem is the fact that liquid petrolatum cannot be metabolized by tissue enzymes as can some of the animal and vegetable oils. It remains a foreign body irritant.

Experiments on animals, as well as necropsy, have demonstrated the gross and microscopic lesions of the lungs which result from the aspiration of oils. These include all varieties, from acute through chronic changes, the latter effects being especially severe. Occasionally infections of the blood stream have taken place, for in almost all instances the oil has carried pathogenic bacteria with it.

The sputum may contain lipophages (large vacuolated mononuclear cells having at times a signet ring appearance). A number of methods are used to identify the oils, but these belong to the field of the chemist and of the pathologist.

The diagnosis of lipid pneumonia will be arrived at by a careful taking of the history, physical examination of the chest, serial roentgenograms, an analysis of the sputum and other laboratory tests. If any surgical intervention is contemplated (as in bronchogenic carcinoma), errors in diagnosis may be serious.

The incidence of lipid pneumonia cannot at present be ascertained with precision, for the clinical types vary greatly, and most of the cases reported have been diagnosed at necropsy. It will be necessary to have a routine histologic examination of the lungs made at necropsy, so that in due time sufficient data may be accumulated to establish correct conclusions.

Every effort should be directed to the prevention of this condition, for it is essentially a man-made disease. This calls for the elimination of every likely cause. Greater care in infant feeding, particularly in the administration of fish oils, will prevent one hazard. It is better to use smaller doses or concentrates. All forcible feeding of cod liver oil and liquid petrolatum must be eliminated. Greater care should be given to the feeding of prematurely born or of weakened and debilitated infants. They should never be fed while lying on their backs. Care in the feeding of comatose persons, the use of gavage and the prevention and control of vomiting will also prevent aspiration pneumonia in a number of cases.

Lipid pneumonia due to intranasal medication offers the most hopeful prospect of prevention, for by the concerted efforts of members of the medical profession, including the otolaryngologists, many cases will be prevented. This is of the utmost importance. Twenty per cent of all the cases of lipid pneumonia thus far reported in the literature have been due to the aspiration of oils into the lungs from either nose drops or sprays.

Cannon concludes his excellent paper with a quotation from Proetz, stressing the importance of the functional point of view in rhinology with the statement that members of the medical profession should take the leadership in an educational campaign to guide the public away from the empiric use of nasal oils for self medication, for . . . "It is likewise progress, though it seldom occurs to us, to abandon old methods . . . when they have been discredited. Not to abandon them after they have been proved harmful is reprehensible."

He makes a plea for the use of aqueous solutions of sodium chloride or of dextrose in preference to the oils.

GORDON, Philadelphia.

CANCER OF THE HEAD AND NECK IN CHILDREN. HAYES E. MARTIN, J. Pediat. **15**:363 (Sept.) 1939.

The most frequent cancer of the head or neck in the children of Martin's series of 44 was retinal glioma. The first symptom of this tumor was dilatation of the pupil, and as the tumor grew it became visible through the dilated pupil as a white reflex, or "cat's eye." The tumor is believed to be congenital, and in over one half of this series the diagnosis was made before the patient reached the age of 2 years. In 75 per cent the disease was unilateral. Treatment depends on the extent of interference with function of the eye. If function of the eye has been

completely destroyed, enucleation is the best treatment when only one eye is involved. When both eyes are involved, enucleation of the eye in which greatest loss of function has occurred should be done, and radiation therapy should be used on the other eye. The next most common location of cancer of the head or neck in children is the region of the nasopharynx, tonsil and soft palate. The most common first symptom in this series was cervical adenopathy or nasal obstruction, or both. In addition to these tumors Martin discusses nasopharyngeal fibroma, myogenic sarcoma of the soft palate, lymphoepithelioma of the tonsil, cancer of the skin of the face, cancer of the thyroid, osteogenic sarcoma, sarcoma of the tongue and papilloma of the larynx.

RAUH, Cincinnati. [AM. J. DIS. CHILD.]

SULFAPYRIDINE IN MENINGOCOCCIC MENINGITIS: REVIEW OF THE LITERATURE AND REPORT OF A CASE. HERMAN I. KANTOR, New York State J. Med. **40**:1526 (Oct. 15) 1940.

A woman aged 66 was admitted to Mount Sinai Hospital, New York, Oct. 10, 1939, in a semiconscious condition. The temperature was 103 F. The respiration was stertorous. The face was flushed and somewhat drawn. There was rigidity of the neck, which was painful on forced flexion. Spinal tap showed cloudy fluid under pressure. The sugar content was 40 mg. per hundred cubic centimeters. A twenty-four hour culture revealed a pure growth of meningococcus. The blood count was 15,800 cells per cubic millimeter, with 93 per cent polymorphonuclears. There was a marked shift to the left. The following routine was adopted: (1) slow intravenous infusion of 5 per cent dextrose in distilled water, with 20 cc. of antimeningococcic serum added every four hours; (2) spinal taps every eight hours for drainage and for the introduction of antimeningococcic serum; (3) sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) in suspension given through a Levine tube into the stomach, with the patient under an oxygen tent; (4) supportive symptomatic therapy as indicated. Within twenty-four hours, the evening of the second day of her illness, the patient was conscious and rational and her temperature was normal. Then nausea appeared, and the administration of sulfapyridine was stopped. The patient became worse, and pneumonia of the upper lobe of the left lung was diagnosed. Sulfapyridine was then given despite the grave condition, and on the fifth day improvement began and continued to recovery. Kantor attributes the successful outcome to sulfapyridine, which has been much used in the treatment of epidemic meningitis abroad but not very widely in the United States.

VOORHEES, New York.

DROPLET INFECTION OF AIR: HIGH-SPEED PHOTOGRAPHY OF DROPLET PRODUCTION BY SNEEZING. M. W. JENNISON and H. E. EDGERTON, Proc. Soc. Exper. Biol. & Med. **43**:455 (March) 1940.

Stroboscopic illumination of high speed photography has been used to study the size and velocity of droplets given off in coughing and sneezing. In the photograph of a man at the downstroke of a sneeze, as presented in the paper, 4,600 particles were counted within the focal range of the camera. Calculations of the movement of the fastest of these droplets showed them to be moving at the rate of 100 feet per second. The number of droplets issuing from the nose is insignificant as compared to the number coming from the mouth. Problems concerning the rate of and distances to evaporation and the minimum size of droplets are being investigated by Jennison and Edgerton.

HANSEN, Minneapolis. [AM. J. DIS. CHILD.]

A PRELIMINARY SURVEY ON THE RELATION OF PHYSICAL DEFECTS TO SCHOLASTIC STANDING. A. C. WOOFTER, West Virginia M. J. **35**:413 (Sept.) 1939.

A study of 360 unselected students was made to ascertain the relation, if any, between physical defects and scholastic class standing and to determine which defects are most detrimental to scholastic progress and should therefore be first chosen for corrective treatment.

It was indicated that infected tonsils caused most days of absence from school and proportionately the greatest lowering of scholastic standing.

Enlarged tonsils stood second to infected tonsils.

Defective vision came next to abnormal conditions of the tonsils in producing a lowering of class standing, and malnutrition followed defective vision.

Because of the small number of cases observed, deafness could not be studied definitely.

SHAWKEY, Charleston, W. Va. [AM. J. DIS. CHILD.]

INTRACRANIAL CAUSES OF HEADACHE. HENRY COHEN, Brit. M. J. **2**:713 (Oct. 7) 1939.

Headaches are usually anatomically classified as intracranial, cranial, reflex, toxic, migraine or psychogenic. The site, duration, character and intensity of a headache will aid in locating the lesion responsible for this symptom. Cohen states that only a relatively small number of sufferers from chronic headache are victims of organic intracranial disease. Headache is a common symptom of tumor of the brain and of cerebral edema, meningeal irritation and diseases of the cerebral arteries. It has been observed that the severity of headache increases with the rapidity of development of the causative lesion and with the definite interference with circulation of the cerebrospinal fluid.

Three varieties of headache are associated with intracranial disease: a dull or throbbing unilaterial pain in the forehead, most often associated with expanding lesions of the brain; a sharp pain in the back of the head, usually seen in cases of subarachnoid hemorrhage and meningitis, and the migrainous type of headache, commonest in vascular lesions.

Various explanations for headaches associated with expanding lesions have been offered. High and low cerebrospinal fluid pressure may be responsible for headache of this type. Excessive tension in the walls of the cerebral blood vessels has been considered a causative factor. Cohen believes that this type of headache is the result of cerebral anoxia.

In the cases of patients complaining chiefly of headache there should be a careful taking of the anamnesis and detailed clinical examination. If an intracranial lesion is suspected, lumbar puncture and encephalographic study should be undertaken. Cohen gives the following suggestions as an aid in diagnosing conditions responsible for headache: Headaches paroxysmal in character, increasing in severity and frequency and accompanied by dizziness, vomiting, mental deterioration, epileptiform attacks or signs of involvement of the facial nerve should suggest organic disease of the brain. Migraine beginning after the age of 40 may be indicative of gross intracranial disease, such as cerebral tumor, syphilis and hypertensive cerebral arteriosclerosis. Unilateral migrainous headache followed by ocular paresis is almost synonymous with the presence of basal intracranial aneurysms. In children severe headache associated with recurring attacks of cyclic vomiting usually indicates a tumor of the posterior fossa. The diagnosis of psychogenic headache should be made only on the basis of positive evidence.

ECHOLS, New Orleans. [ARCH. NEUROL. & PSYCHIAT.]

SOME FACTORS INFLUENCING THE SPREAD OF SCARLET FEVER IN AN INSTITUTION. E. V. KEOGH, IAN MACDONALD, JOAN BATTLE, R. T. SIMONS and STANLEY WILLIAMS, J. Hyg. **39**:664 (Nov.) 1939.

In an outbreak of scarlet fever due to Streptococcus pyogenes type II, in Melbourne (Victoria, Australia), 59 children in a single ward were studied. Material from the throat was taken by swab weekly for ten weeks and again after an interval of thirteen weeks. In 16 patients scarlet fever with a rash developed, and in 23 the disease appeared without a rash (infection with Str. pyogenes type II).

The authors state in their summary: "Haemolytic streptococci were isolated at least once from all but four children.

"Seven group A strains and one group C strain were present in the ward. Two group A strains and the group C strain spread freely, but were not associated with scarlet fever or any evident clinical symptoms. . .

"Children with and without tonsils were equally susceptible to infection with haemolytic streptococci, irrespective of group or type, but the infections persisted much longer in the children with tonsils.

"Sulphanilamide therapy did not check the epidemic."

JANNEY, Wauwatosa, Wis. [AM. J. DIS. CHILD.]

TONSILLECTOMY AND NEPHRITIS OF CHILDHOOD. R. S. ILLINGWORTH, Lancet **2**:1013 (Nov. 11) 1939.

Opinions on the value of tonsillectomy in cases of nephritis are many and varied.

Of 301 patients admitted to the Hospital for Sick Children, London, England, in the last eleven years for acute nephritis, 61 (20.2 per cent) had undergone tonsillectomy some months or years previously.

The conclusion reached is that tonsillectomy does not prevent nephritis but may predispose to it, that it does not cure nephritis or prevent it from progressing to a chronic stage and that it may cause nephritis.

LANGMANN, New York. [AM. J. DIS. CHILD.]

COD LIVER OIL DRESSINGS: THEIR MODE OF ACTION. M. LICHTENSTEIN, Lancet **2**:1023 (Nov. 11) 1939.

The local treatment of infected wounds, burns, ulcers, amputation stumps, osteomyelitis, etc., with dressings soaked in cod liver oil or covered with a thick layer of ointment containing a large amount (25 per cent) of cod liver oil in a neutral base (petrolatum) has come into prominence since the publication of Löhr in 1934.

The striking results obtained by many authors with cod liver oil dressings are due, in some degree at least, to the bactericidal power of this substance. Irradiation of cod liver oil enhances this power. Furthermore, the oil is nonirritating to the tissues and supplies them with vitamins.

LANGMANN, New York. [AM. J. DIS. CHILD.]

INFLAMMATORY DISEASES OF THE UPPER PORTION OF THE RESPIRATORY TRACT. G. HALLORAN, M. J. Australia **1**:182 (Feb. 10) 1940.

Nasal obstruction is a preliminary factor in inflammatory diseases of the nose and sinuses. Halloran considers whether a typically narrow face in which nasal disorders are prone to develop is not already becoming characteristic of white Australians. Some tropical environments and the prevalence of sea bathing may also tend to increase sinal disease. Repeated head colds are undoubtedly the commonest cause of chronic sinusitis.

Antrums are present at birth, but the ethmoids must also be considered possible foci of infection. In diagnosis, transillumination must not be relied on. Diagnostic puncture and roentgen examination offer the most effective means of diagnosis.

In treatment, ventilation of the nose is essential. For chronic sinusitis radical operation is not advocated, but puncture of the antrum is frequently used.

Infection of tonsils and adenoids undoubtedly accompanies chronic sinusitis, but removal of these structures does not always hasten recovery.

There is apparently no definite relation between sinal infection and mental disease. There is definite relation between infection of the tonsils and cultures persistently yielding the streptococcus.

TENNEY, Madison, Wis. [AM. J. DIS. CHILD.]

TETANUS SECONDARY TO A NASAL LESION. DONALD PATERSON, Proc. Roy. Soc. Med. **33**:373 (May) 1940.

A boy aged 2½ years was admitted to the hospital because he had suffered from convulsions for four days. On physical examination the only abnormality found was paralysis of the left facial nerve, and it was thought at the time that there must have been some cerebral damage at birth or a recent vascular lesion and that the fits were epileptic. Three days later the temperature began to rise, and next day well marked trismus was noticed. The child presented a typical picture of tetanus. He was treated with antitetanic serum and avertin with amylene hydrate and was fed through the nasal tube entirely. When the nasal tube was passed up the left naris, the naris was found to be blocked with three lumps of coal as large as peas. The child recovered in four weeks from the onset of the illness.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

BILATERAL OPHTHALMOPLEGIA FROM INTRACRANIAL EXTENSION OF A TUMOR OF THE RIGHT MAXILLARY SINUS. MARIA ANDRÉ, J. belge de neurol. et de psychiat. **40**:21 (Jan.) 1940.

André reports the case of a man aged 57 with carcinoma of the right maxillary sinus. During his illness headaches and bilateral ptosis developed. Later, examination revealed the presence of bilateral total ophthalmoplegia, corneal anesthesia, slight diminution of visual acuity and moderate atrophy of the optic nerves but no papilledema or palpebral edema. Roentgenograms showed extensive destruction of the sphenoid bone. The author reports this case because of the rarity of the syndrome of bilateral ophthalmoplegia in patients with tumor of the maxillary sinus or cancer of the nose.

DE JONG, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

PSYCHIC TREATMENT OF STUTTERING AND PHONASTHENIA. A. TAMM, Acta pædiat. **25**:277, 1939.

The clinical features of stuttering clearly characterize it as a neurotic symptom; in some cases stuttering symbolizes hysteria and in others a compulsion neurosis. Like other compulsions, it is observed oftener in men than in women. The curability of the symptom is not directly proportional to the severity of the speech impediment; "the stutterer who does not stutter"—that is, the one who elaborately avoids pronouncing the troublesome words—is often more difficult to help than the one who tackles the difficulties, no matter how unsuccessfully from the point of view of effective speech. The stutterer's past is apt to be characterized by repressions and his present by ambivalence. Masturbation and autoerotic tendencies are fruitful sources of trouble. Sometimes speech is not involved and only the writing is affected. Certain letters that refer directly or indirectly to subjects that are socially taboo cause more difficulties than neutral letters. The symptom is peculiar to childhood but sometimes does not appear until adult life. Organic insults can accelerate its arrival. A lack of correlation between the development of thought and of speech may bring it out. Two types of treatment prevail; the first is simply supervised practice in phonation, and the second seeks by psychiatric or psychoanalytic methods to uncover the cause of the disturbance and to obviate it.

Phonasthenia is defined as incapacitating vocal fatigue without proportionate anatomic cause; it may be due to constitutional disturbances but is often a symptom of neurosis. It is peculiarly apt to overtake persons who use their voices a great deal. It is commoner in women than in men. Its background is like that of stuttering. The treatment is largely psychiatric, although physical modalities are not to be despised.

MCCUNE, New York. [AM. J. DIS. CHILD.]

Society Transactions

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, MIDDLE SECTION, AND CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Joint Meeting, Jan. 27, 1941

An Evaluation of the Ménière Syndrome. Dr. W. E. Grove.

In 1861, almost seventy years ago, Ménière described a group of symptoms which he believed indicative of a separate disease entity. From the pathologic description of one of his cases, which came to autopsy, it would appear that in that particular case he was dealing with purulent labyrinthitis. His original description of the Ménière syndrome was classic and left little to be added by future clinicians.

He described the sudden attack of vertigo without any apparent preceding illness, the recurrence of the attacks, the extreme prostration of the patient and the tendency of the attack to recur. He observed that tinnitus preceded the first attack, persisted between attacks, was often increased during the attack and was not influenced by pressure on the carotid. He noted the impaired hearing in one and sometimes in both ears, which began with the tinnitus and progressed to complete deafness without evidence of any ear disease. In a series of papers he described 9 such cases.

From the foregoing statements it is clear that Ménière had a clear conception of the syndrome which now bears his name and that little has been added to the symptomatic picture of the condition since his time.

The factors described by various authors as making up the cause of the syndrome are legion, but, according to most of them, the probable location of the lesion is the cochleovestibular apparatus, either in the nerve itself or in its peripheral distribution or in its central connection.

Mygind and Dederding expressed the opinion that the symptoms were caused by abnormal water metabolism resulting in retention of fluid in the labyrinth and in other portions of the body from which symptoms were emanating, because these symptoms were relieved by dehydration produced by medication with pilocarpine and limitation of fluid intake.

Furstenberg concluded that Mygind and Dederding were in error in ascribing a disturbed water metabolism as the cause, for the reason that in eliminating large quantities of water by perspiration and diuresis Mygind and Dederding were also eliminating electrolyte. He expressed the opinion that the relief of symptoms came from the elimination of sodium stored in the body.

Dandy, McKenzie, Adson and others called attention to abnormal arterial loops from the internal auditory artery or the inferior cerebellar artery surrounding and in contact with the auditory nerve; they expressed the opinion that these arterial loops may have been a factor in the production of symptoms in at least some of their cases. In this connection Torkildson found at operation an abnormal artery about the size of the facial nerve passing into the internal auditory meatus along with the acoustic and facial nerves. Removal of the artery alone was followed by a remission of the syndrome for four months, i. e., up to the time of the publication of his report.

If such arterial loops cause the symptoms of Ménière's disease, they must operate by pressure on the nerve. Crowe, Furstenberg and Dandy examined these nerves microscopically and found them normal. Furthermore, as Crowe pointed

out, such arterial loops could damage the nerve only if they showed arteriosclerotic changes, and cerebral arteriosclerosis is extremely rare in the entire group of reported cases of the Ménière syndrome. He makes the further point that if these arterial loops were causing damage to the acoustic nerve within the confines of the internal auditory meatus, one would also expect damage to the facial nerve, which is never found in this condition.

That allergy plays some role in causing the Ménière syndrome is apparent from the reports of various observers showing that the Ménière syndrome frequently appears in patients with other allergic manifestations, that the withdrawal of the offending allergens will cause cessation of the attacks and that readministration of the allergens will again produce an attack.

Vasomotor instability and increased capillary permeability are features of an allergic condition. If such an allergic reaction can alter water metabolism and produce localized edema of tissues or change the mineral metabolism with the same localized edema, then the causation of the Ménière syndrome could be logically explained on an allergic basis in many cases.

The localization of the trouble is not in the nerve alone, because no pathologic condition has ever been found there microscopically. The pathologic alteration is not exclusively in the end organ, because section of the acoustic nerve does not abolish the tinnitus, although it cures the vertigo. However, a biochemical process, whatever its cause, operating both in the end organ and in the basal nuclei and producing extracellular edema in both regions because of loss of vasomotor stability and increase of capillary permeability could adequately explain all the aural manifestations of the syndrome.

Until Hallpike described the temporal bones of 2 persons who died following intracranial section of the vestibular nerve, there were no postmortem data about this interesting condition. His sections showed gross dilatation of the entire endolymph system. Reissner's membrane was pushed against and adhered to the walls of the cochlea, thus obliterating the scala vestibuli. The tectorial membrane was flattened down on the organ of Corti, which showed marked degenerative changes. There were also degenerative changes in the sensory epithelium of the vestibule and of the stria vascularis. Whether this condition was due to oversecretion of endolymph or to faulty absorption of this fluid he could not tell, but he assumed it was due to the latter, because an absence of perisaccular connective tissue was noted.

He explained the individual attacks of vertigo as being initiated by bouts of asphyxia of the labyrinthine end organs, brought about by extremely rapid rises of fluid pressure in response to relatively small increases of endolymph. Before this can happen, however, the cochlear duct must first have dilated to the limits of its bony walls.

The symptoms of the Ménière syndrome are so characteristic and so well known that I will not describe them here. It is a condition of adult life; most of the patients are in the third, fourth and fifth decades. However, the condition has been described in patients at the ages of 3, 6 and 14.

It usually begins with a sudden violent attack of vertigo. However, in a few cases the first symptom may be unilateral deafness or tinnitus. The vertigo differs in its intensity from that produced by any other intracranial condition, such as acoustic neuroma, tumor of the pontile angle, a degenerative lesion or the like. The vertigo occasioned by labyrinthitis is just as severe as that of a Ménière attack, but after it once subsides, and the labyrinth is dead, it does not recur.

Nystagmus may, but does not always, accompany the attack, and most investigators are of the opinion that the caloric responses are of no value in making a diagnosis or in lateralizing the condition.

Deafness usually follows but may precede the initial attack. It is usually unilateral but may be bilateral in as high as 38 per cent of the cases. It is of an inner ear or perceptive type and is progressive.

There is nothing about the tinnitus to distinguish it from that arising from otosclerosis or any other lesion of the ear. If, as Mygind stated, it is due to an intracellular edema in the organ of Corti it should disappear in every case after section of the nerve, but it does not. I would rather believe it was due to a similar concomitant process operating in the end organ and the cochlear nuclei. Such a process, possibly edema, in the cochlear nuclei would explain the persistence of tinnitus after section of the eighth nerve.

The Ménière attack is not usually preceded by any prodromal symptoms, but Crowe found the following aura in 13 per cent of his cases: increased tinnitus, fulness in the head, faintness and nausea, increased deafness and tinnitus which improved after the attack, burning sensation in the throat and tingling and numbness in the fingers of one hand.

Spontaneous remissions of vertigo and tinnitus occur with this syndrome for no particular reason. It is questionable, therefore, if one can speak of a cure of the condition. As such remissions are also known in allergic states, it strengthens my opinion that allergy plays some role in the causation of this symptom complex.

The apoplectiform disabling attack of vertigo which recurs from time to time accompanied by tinnitus and progressive deafness makes the diagnosis of the condition simple.

That brings me to the discussion of a pseudo Ménière syndrome. In the true Ménière syndrome the attack of vertigo is the first symptom in a large majority of cases. In some the tinnitus and deafness soon follow, in others these symptoms are delayed in their appearance for long periods, and in a few the tinnitus and deafness never occur. It is quite possible that these are all gradations of the same condition; if the attack of vertigo is characteristically sudden and severe with a definite sense of rotation present and if it disappears only to recur again, I believe the condition should be referred to as a Ménière syndrome even in the absence of tinnitus and deafness.

On the other hand, the otologist sees many patients with unexplainable tinnitus and deafness. It is also possible that some of the complexes observed are way stations in the development of a true Ménière syndrome and that, if the otologist observes them long enough, the vertigo may appear in some of them.

In the past, treatment of the Ménière syndrome has been on a purely empiric basis. At the present time the treatment is either medical or surgical. The management of the condition, as outlined by Mygind and Dederding, is based on a restricted fluid intake, a diet low in salt for the purpose of increasing diuresis, a reduction diet for patients who are overweight, together with exercises, massage and ultraviolet ray therapy to stimulate vasomotor tone. Dederding reported a disappearance of the vertigo and nystagmus in all but 7 of a series of 157 cases, and an improvement of the hearing in all but 6.

Furstenberg, Lashmet and Lathrop noted that the retention of water and sodium produced the attack while the retention of water without sodium did not and, conversely, the loss of water with retention of sodium produced an attack while the loss of water and sodium did not. Furstenberg and his co-workers, therefore, centered their attack on the sodium factor by restricting the intake of sodium and increasing the elimination of this element by the use of ammonium chloride and allowing their patients all the fluid desired. They reported favorable results with this regimen, as did Walsh and Adson, Brown, Bartels and others. Walsh and Adson substituted potassium nitrate for ammonium chloride as a sodium-eliminating drug.

All those employing the medical treatment call attention to the difficulty experienced in keeping patients who have returned home on a medical regimen. It is doubtful, therefore, if one can speak of a cure of this condition under a medical regimen, knowing that long periods of remission in the symptoms of this complex occur without any known cause.

Sheldon and Horton called attention to the quick relief of symptoms after the intravenous injection of histamine acid phosphate, which one of them had been

using in the treatment of a vascular type of headache, and stated that several patients improved under this treatment who had not improved under the Furstenberg regimen.

The surgical treatment of the Ménière syndrome aims at an interruption of impulses arising within the labyrinth and passing to the brain. It is predicated on the assumption that the lesion causing the symptoms is located either in the labyrinth or in the acoustic nerve. This surgical intervention consists either in severing the eighth nerve or its vestibular portion intracranially or in obliterating the end organs in the labyrinth.

This operation, designed by Krause and previously used by Frazier and Ballance, has been successfully used by Dandy in more than 250 cases without a fatality. If the hearing is bad, he sections the whole nerve; if relatively good, only the vestibular portion.

McKenzie and McGregor have shown that while the separation of the cochlear and vestibular fibers is fairly complete after the acoustic nerve leaves the internal auditory meatus, there is some intermingling of these fibers along the line of separation. Dandy, however, pointed out that quite a large number of cochlear fibers can be severed during the differential section of the vestibular nerve without further damage of the hearing. If the higher tones have not already been lost owing to the disease, it is the high tones which are damaged by a partial section of the cochlear nerve. Dandy had no mortality from section of the eighth nerve, but McKenzie reported one fatality and Hallpike two. The postoperative complications reported by Dandy were as follows: transient facial paralysis, transient diplopia, transient nystagmus, transient postoperative vertigo. In regard to the hearing after differential section of the vestibular nerve, Crowe reported that of 72 patients on whom Dandy had performed a section of the vestibular nerve, the deafness was worse in 22, unchanged in 36 and improved in 14.

While section of the eighth nerve or of its vestibular component offers great possibilities of relief for the intractable and disabling vertigo, the same cannot be said as to the tinnitus; this is admitted by all neurosurgeons doing this type of operation. This fact alone would argue against the exclusive localization of the lesion in the labyrinth.

In recent years, otologists, following the same line of reasoning as exponents of nerve section, namely, that it is necessary to interrupt the abnormal impulses from the labyrinth, have been practicing the destruction of the sensory epithelium in the end organ; Mollison, by injecting absolute alcohol into the external semicircular canal, Berggren, by injecting it through the promontory, and Putnam, by drilling into the superior canal and using coagulation. Naturally, this sort of treatment can be used only when the hearing is almost entirely gone.

The Ménière syndrome is a fairly common condition affecting middle life characterized by a triad of symptoms, vertigo, tinnitus and deafness, of unkown cause and little known pathologic nature. In assigning the cause, more serious consideration must be given to allergy, avitaminosis and disturbances in the endocrine balance than hitherto.

If the pathologic condition of the temporal bone described by Hallpike is characteristic of every case, then the main pathologic feature of the condition is a waterlogging or edema, as was maintained by Mygind, Dederding, Furstenberg and others. This condition is probably due to a metabolic disturbance, but whether a disturbance in the water balance, as Mygind and Dederding maintained, or a disturbance in the metabolism of the sodium ion, as Furstenberg stated, or both, is not entirely clear.

The operation of this disturbance in metabolism seems to find its greatest expression in the labyrinth, where it initiates attacks of vertigo, deafness and, probably, some of the tinnitus. That not all of the tinnitus originates in the end organ is evidenced by the fact that some persists in many cases after total destruction of the labyrinth or a severance of the nerves to this organ. May it not be

that this same waterlogging process is operating both in the end organ and also in the central cochlear nuclei?

The success of the dehydration treatment of Mygind and Dederding and of the sodium elimination treatment of Furstenberg, as well as of the more recent treatment with histamine when carried out under proper hospital supervision, suggests that the patient should be given the opportunity of trying medical treatment before submitting to surgical intervention.

Surgical intervention should be reserved for those patients who have not responded to a medical regimen, for those patients who for economic or other reasons cannot be kept on a medical regimen and for those patients whose occupations are such as to preclude the possibility of any return of vertigo because of carelessness in following a medical regimen. Of the surgical measures proposed, it would seem that total section of the acoustic nerve is indicated for those whose hearing in the affected ear has fallen below a usable level and that the differential section of the vestibular portion of the nerve is the operation of choice for those with usable hearing. These operations should not prove hazardous in the hands of the competent neurosurgeon.

DISCUSSION

DR. T. ROY GITTINS, Sioux City, Iowa: Dr. Grove has covered the subject of Ménière's syndrome in the complete manner characteristic of all his writings. I am glad that he emphasized how little is really known concerning the nature and location of the pathologic condition responsible for this syndrome and also that many of the symptoms are more suggestive of involvement of cortical or subcortical areas in the brain than of a lesion of the inner ear. He also stressed the point that allergy should be given more consideration as a causative factor in this syndrome. I have followed the literature carefully for many years in relation to the subject of vertigo and associated symptoms and have found that Dr. Grove is one of the few otologists who have given much attention to allergic or vasomotor possibilities in the production of this syndrome.

In the short time at my disposal in this discussion I wish to stress the characteristics and prevalence of what I choose to call, for want of a better name, the vasomotor type. An allergic or vasomotor reaction, possibly of a biochemical nature, in the tissues of the inner ear, the auditory nerve or the brain tissue would naturally be a transient upset rather than a pathologic process. History, then, must take precedence over operative or postmortem evidence in the differential diagnosis of everyday practice.

Attacks of real rotary vertigo with pallor, cold perspiration, nausea and vomiting may appear as prodromal symptoms in an attack of migraine. What is of more importance, however, this symptom complex replaces the entire migraine or vasomotor syndrome in many patients for months or years. Such attacks are typical of the classic Ménière syndrome, except that tinnitus and loss of hearing are usually not present. These attacks, because of their short duration and lack of after-effects, though they are common, are seldom properly diagnosed. Rarely does an otologist see such patients during their attacks, and rarely does the general physician, who does see them, consider the real nature of the disturbance. The diagnosis is usually heart trouble, acute indigestion or food poisoning. Because the appearance of the patient and the gastrointestinal symptoms, especially vomiting, are dramatic, little attention is given to the causative vertigo by the patient or the physician. As a rule, the general physician or internist does not need to give much thought to the actual nature of the disturbances, because the attacks are usually of short duration, and the patient may be up and around as well as ever in an hour or a day. Occasionally, the attacks occur at intervals for a week or more, especially on sudden changes of position. The usual patient recovers quickly, regardless of, and usually in spite of, the treatment advised.

I have seen but few patients during their attacks but have heard of a great many attacks of this nature while taking routine histories from patients who come to the office because of periodic headache or neuralgia of the vasomotor type. In only 3 instances have I seen rotary nystagmus; but in these instances it was typical of irritative labyrinthitis during the short time it was present. For over two years, vertiginous attacks of short duration with pallor, cold perspiration and nausea replaced my own migraine syndrome; and I assure you that a patient suffering from this condition has reason to be seriously disturbed and anxious to be assured that there is probably no real pathologic condition in the inner ear or brain. Little has been said in the literature concerning this type of Ménière syndrome (or possibly pseudo Ménière syndrome), but I feel, after interviewing patients with periodic vasomotor headaches for many years, that it is more common than any other form of vestibular disturbance. Occasionally, of course, a patient may have chronic progressive deafness and tinnitus coincidentally with a tendency to vasomotor upsets. In such instances, the differential diagnosis is naturally a real problem, and time may be the most important factor in the final decision.

In my experience, it is rare to obtain a history of vertiginous attacks from the really allergic person, such as the sufferer from hay fever or asthma. It is not rare, however, to obtain such a history from the person who has suffered periodically for years with headaches or neuralgia of the migrainous or vasomotor type. Whether or not migraine and other types of periodic vasomotor headache usually have an allergic basis, I still think is open to serious question, notwithstanding some enthusiastic reports in the literature, especially in relation to food sensitivity.

Dr. Grove has stressed the important point of spontaneous remissions in Ménière's syndrome, and certainly the vasomotor type which I am discussing has the same tendency to spontaneous relief as all types of vasomotor disturbances, especially headache and neuralgia. The results from treatment of any of these conditions should be evaluated with the thought in mind that spontaneous recoveries from any syndromes, at least in my own experience, are more common in the person with vasomotor instability than recoveries after all treatments and operations combined. In my limited experience, the results with low sodium and water intake and ammonium chloride therapy have been poor in persons with the vasomotor type of this syndrome; much poorer than in those with the more classic type, which probably relates to a real lesion in the inner ear or in the auditory nerve.

Finally, I wish to stress again my belief in the great prevalence of vertiginous attacks with associated symptoms which are probably of vasomotor and transient origin. I also want to stress that otologists will not necessarily realize how great is this prevalence unless they carefully inquire into the histories of the patients who come to them because of periodic headache or neuralgia. These patients rarely volunteer any history relative to the vertiginous attacks because they have felt and in most instances, have been told, that such disturbances were in the domain of the general physician or internist because of the gastrointestinal and other general symptoms.

I am glad to say that I have enjoyed reading, studying and hearing Dr. Grove's paper. I also wish to express my appreciation of the opportunity to enter into the discussion of this intriguing disease, syndrome or pseudosyndrome.

Concerning the symptomatic picture, little has been added through these many years since the classic treatise by Ménière. Likewise, concerning the actual nature and location of the process, be it biochemical or pathologic, which accounts for the symptoms, little has been added through these same years, except possibly in a negative way.

Dr. John Shea, Memphis, Tenn.: I think all have enjoyed Dr. Grove's presentation of his subject. I merely wish to add a little about histamine therapy. During acute attacks, when the patient is bedridden, histamine will relieve him

more quickly than anything else I have tried. The patient may be tested by giving 0.5 cc. of histamine diphosphate; if there is no reaction, the dose may be increased to 1 mg. It may be repeated in two, three or four days if necessary, and then one must give a sustaining dose of 0.5 cc. twice a week. This year at the meeting of the American College of Surgeons, Dr. Bayard T. Horton of the Mayo Clinic compared histamine therapy with surgical intervention. Since surgery can offer cures in only 75 per cent of the cases and while histamine is so satisfactory, the latter should first be given a worthy trial.

Dr. Harold I. Lillie, Rochester, Minn.: Dr. Hunter Sheldon observed that 1 patient, in addition to having been relieved of a histaminic headache, was relieved also of the vertigo of which she complained when histamine had been administered intravenously. I think this group of patients is linked with vasomotor syndromes suggestive of an allergic causative factor. Some patients do well when histaminase (torantil, a water-soluble pulverized protein prepared from the mucous membrane of the small intestines) is administered. It is not felt that the treatment is curative; it is really the treatment for an attack, and if maintenance treatment is carried out, as Dr. Shea said, the probabilities are that these patients will be relieved.

Another treatment was suggested by an old practitioner. He told me that if he gave 1/75 or 1/60 grain (0.0009 or 0.0013 Gm.) of atropine sulfate, most patients suffering from acute attacks of vertigo would be relieved. That probably is explained on the basis of an effect on the vasomotor system, and it is probable that that type of vertiginous attack is caused by some disturbance of the vascular system that atropine overcomes. I have a great deal of respect for the knowledge of practical therapeutic measures that general practitioners possess.

There are toxic types of vertigo. One from which I suffer, especially if I am tired, is the cumulative effect of smoking. I have found that it is usually relieved by taking about 2 fluid ounces (60 cc.) of Bourbon whisky.

Portmann's operation has not been mentioned as a method of treatment for vertigo. I recently saw a patient on whom that operation was performed; it was followed by complete relief.

Physicians are on the threshold of learning more about vertigo and its causative factors. I would not subject myself to an operation on the eighth nerve to relieve vertigo until all other methods of treatment had been tried, although I have seen several dramatic results from that treatment. Complete physical and laboratory examinations must be made of every patient who complains of vertigo in order that the general condition may be known before any method of treatment is instituted.

Dr. Harry W. Lyman, St. Louis: I will not take any time in complimenting Dr. Grove. It seems to me an unfortunate thing that Ménière's name has been attached so firmly to this symptom of vertigo. Of course there is the triad—deafness, tinnitus and vertigo—but there are many who when they see a patient with vertigo immediately say "Ménière syndrome or pseudo Ménière syndrome" and think they have made a diagnosis. It has been brought out that there are probably a number of underlying causes for the disturbance of hearing. I think the most striking one is the allergic condition. As Dr. Grove mentioned, the symptoms can be brought on by administering the offending agent. The same symptoms are seen with a tumor of the eighth nerve or one of the pontile angle, and on superficial observation the physician might not be able to distinguish one from the other. In other cases the syndrome is due to toxic intoxication of the labyrinth. I recall cases in which the patients presented deafness, tinnitus and vertigo. One patient was sent in with a diagnosis of Ménière's disease; it was disclosed later that the deafness was of eighteen years' standing and due to mumps, and the vertigo and tinnitus were of one year's duration. I think one must get away from the idea that one is dealing with a single disease. There are, I think, a number of underlying causes, and each case must be studied carefully in an effort to determine the origin. As to surgical relief, I think most otologists agree

it should be a last resort. That is, medical treatment and careful search for underlying causes should be undertaken first. Of course, in many cases the vertigo is so disabling that the patient is willing and even anxious to submit to any operation that relieves it. Operation does relieve it, and the patient does not mind the deafness, but operation should not be done in cases in which attacks are brought on by such things as eating chocolate.

Dr. Millard F. Arbuckle, St. Louis: I have heard emphasis laid on what is called horse sense; the use of horse sense is essential in the study of these patients. I have found it helpful to have them studied by a good internist from the standpoint of disease of the circulatory system or of the endocrine system. I have seen quite a few patients with the complaints mentioned in this worth while paper in whom disorder of the thyroid gland, particularly hypothyroidism, was present, and in whom the symptoms were relieved by the administration of thyroid gland. Then I had one experience with a celebrated physician who woke up one morning so dizzy he could not get out of bed and was wholly disabled for a week. Finally, after two weeks or so, he made a complete recovery by taking vitamin B with no other treatment.

Dr. Thomas Galloway, Evanston, Ill.: I should like to ask Dr. Grove whether he used thiamine hydrochloride (vitamin B_1) intravenously.

Dr. W. E. Grove, Milwaukee: This paper was in the nature of an evaluation and not the result of personal experience with this condition; there is not much to add except to thank the discussers. In reply to Dr. Galloway, I have not used thiamine hydrochloride, but I should like to say something more about histamine. In the paper due credit is given Drs. Sheldon and Horton for their work with this agent. I have had considerable success with it, particularly in the acute period of vertigo. As Dr. Shea said, the drug should be given slowly and intravenously. I have used the amount originally prescribed by Sheldon and Horton (1.9 mg. dissolved in 250 cc. of physiologic solution of sodium chloride) and injected it over a period of one and one-half to two hours. I have also had some experience with the follow-up treatment in which the drug is given subcutaneously in smaller doses two or three times a week. Heartened a little by the work of Sheldon and Horton, and feeling, as I do, that some of the headache and dizziness which come on in spells after a head injury is possibly and probably due to vasomotor disturbance at that time, I have made use of the drug in that type of case and have had some success. The success has not been apparent in every case, but in some the results were rather dramatic.

Complications of Esophageal Perforations. Dr. Paul H. Holinger.

A brief review of the anatomy of the esophagus and surrounding structures is presented, and the causation of esophageal perforations and their complications is discussed.

The complications of esophageal perforations are: (1) infections—periesophagitis and abscess; (2) hemorrhage; (3) perforations of the pleura; (4) emphysema; (5) pericarditis; (6) perforations of the trachea and bronchi.

The diagnosis and treatment of these complications are discussed, and representative cases are presented.

DISCUSSION

Dr. Samuel J. Pearlman, Chicago: I find myself very much in the position of Dr. Brunner: This paper is remarkably complete, and I cannot add anything to it. I agree with the essayist, so I cannot give you the spectacle of a lively debate. It would not be possible, of course, to cover the entire paper and take up the various details. I should like, if I may, to throw some light on the subject by discussing my own fears and anxieties in situations of this character and to limit the discussion to the narrow aspect that most concerns laryngologists, leaving the difficulties that occur in the chest to thoracic surgeons.

A review of the literature shows that the thoracic surgeon is inclined to favor surgical intervention when the difficulty is in the neck, advising the laryngologist to operate and not hesitating to operate himself; but he displays a laudable caution when the trouble is in the chest. Without casting aspersions, is it possible that the attitude here is influenced chiefly not by the danger of the disease process but by the technical difficulties of the approach?

Now, as to perforations high in the neck, I do not know how other laryngologists are affected by them, but for my part I am always anxious. Given a foreign body that may or may not have perforated, how can one tell? If I have removed it, have I done any damage in removing it? If I avoid operating and mediastinitis rapidly develops, my conscience plagues me. I say to myself, had I operated early, this might not have happened. On the other hand, if I want to intervene and the patient refuses, and a colleague bolsters him up in his refusal, and nothing develops or a localized periesophageal abscess evacuates itself spontaneously, I am in the position of having advocated something that was not necessary for a condition which cured itself.

In the literature there are reports of any number of astounding cases with a great deal of trauma to the esophagus in which the patients recovered without external intervention. I remember a case reported by Dr. Iglauer and another from the Mayo Clinic in which a lead toy soldier went through the esophagus and into the mediastinum. This was removed by esophagoscopy, and complete recovery ensued. I remember in my own experience a patient who swallowed a chicken bone, which disappeared after a probang had been used. The bone disappeared but the patient's discomfort continued. On esophagoscopy nothing could be seen at first. I was prepared to quit, when a white spot, the size of a match head, showed itself just below the pinchcock; seized with a forceps, out came this bone as long as a match. That patient made a complete recovery although an infected foreign body was outside and parallel to his esophagus for several days. I do not know the complete answer to the problem presented by this type of case. It is difficult to get a clear answer from the literature. It does seem that recently there is a little more tendency to wait awhile. This does not mean that surgical intervention has not its proper time and place. It is obvious, however, that there can be no absolute rules to follow, much as one would like to have such rules. One's guide is the best of skill and judgment one can muster, with the knowledge that all one's experience may not avail.

Dr Samuel Iglauer, Cincinnati: Dr. Holinger has raised an important question, namely, when to operate. There is no argument about the cause of these complications, and there is little more to be said about diagnosis, except that roentgenologic examination will often enable one to make a diagnosis of perforation before it can be made by any other method; i. e., roentgenograms taken early will often show gas bubbles about the foreign body when there is no palpable emphysema in the neck. When one suspects perforation and wants to take roentgenograms, I think it better to use iodized poppyseed oil 40 per cent than barium sulfate, because if there is any leakage or the trachea is involved, the iodized oil is less irritating.

Dr. Pearlman has raised the same question about when to operate. That reminds me of the treatment of appendicitis in the early days. The procedure used to be to give morphine and wait. That is not done now. The appendix is taken out, often before the diagnosis is made. There is talk of the policy of watchful waiting. What is the waiting for? It is waiting to see whether the patient is going to get better or get worse. The longer the wait, the worse the patient becomes; the longer the wait, the less chance for success. The surgical approach in the neck is not difficult. Suppose one gets down to the esophagus, and there is no pus; one has done no particular harm. On the other hand, if spreading mediastinitis or pleurisy develops, it may be too late. Therefore, I

think it better to take a chance with operation than to delay. I think Dr. Holinger was clever when he approached the foreign body through the neck and then, instead of pulling it out, pushed it back into the esophagoscope.

I do not blame the thoracic surgeon for being timid about posterior mediastinotomy. If he ruptures the pleura, complications occur. On the whole, I should say he would be much safer in operating than in waiting.

DR. MILLARD F. ARBUCKLE, St. Louis: I have been delighted with Dr. Holinger's presentation and with the discussion of this important subject. Dr. Holinger's opening statement was that these cases are "notorious for the amount of trouble they cause." I think all will agree with that. Before I forget it, Dr. Iglauer mentioned gas bubbles. It must be remembered that often there is a gas bacillus infection, which is deadly under the best of conditions.

These patients, it seems to me, might be divided into two classes, the ones who are so indigent that they are inclined to let nature take its course and the well to do group. Those in the latter group are much more likely to die of mediastinitis than those in the toughened first group. I remember a doctor who had been eating rabbit and who got a bone in his esophagus and promptly died of mediastinitis. The next patient with this complication I saw was a drunken quarry worker. He had a chicken rib through the wall of his esophagus into the mediastinum, with all the symptoms of mediastinal infection. When it was taken out he got up and left the hospital under protest. Not long ago I saw a steel worker who had half a wishbone through the esophageal wall into the mediastinum. I pulled it out, and he got well.

In deciding on treatment for esophageal injury one must consider the nature and extent of the injury. It is well recognized that such injuries may range from slight laceration (as is almost certainly true in every foreign body case) to more severe injury, such as a small puncture wound, or to still more severe injury, such as an extensive tear or rupture of the esophagus, with pneumothorax. Patients with slight laceration usually get well with only rest of the esophagus. Those with more severe injuries get well in many instances with similar treatment, but I am always more apprehensive about them for the succeeding day or two, until I am sure they do not show signs of mediastinitis. Chemotherapy is an important part of their treatment, I think, at least as a prophylactic measure. For those patients in whom one is sure there is a tear or rupture of the esophagus, immediate drainage of the mediastinum is indicated—if in the upper part of the mediastinum, cervical drainage, and if low down along the course of the esophagus, drainage by the posterior route.

I feel strongly that watchful waiting for the development of proof of mediastinitis or abscess formation deprives the patient with this extensive type of injury of his best chance for recovery.

DR. THOMAS GALLOWAY, Evanston, Ill.: I rise to record my vote in favor of surgical intervention after perforation. I was surprised to hear Dr. Holinger say that most men believe that one should wait; it was my impression that the consensus was quite otherwise. In the first place, when one goes down only to the esophagus in the absence of infection, one does no harm. In work on the upper part of the esophagus in the dog, if I put in a preliminary pack, there was no descending infection and I could do almost anything afterward in that area. I should think in any case in which one is certain of perforation in a region which is accessible through the neck, one would open to the perforation and pack it off with external drainage.

Cervical mediastinotomy in a clean field has little element of danger; perforation with infection without external drainage has the major odds against recovery.

DR. PAUL H. HOLINGER: This discussion has certainly been stimulating and demonstrates the importance of open discussion on such a controversial subject. I believe I must clarify a point which is covered fully in the paper but which because of the limited time could be covered only hastily. I agree that immediate

mediastinotomy is indicated in cases of mediastinitis and extending periesophageal suppuration. Dr. Arbuckle demonstrated that beautifully in the cases he presented. My mention of conservatism pertained to patients in whose cases the diagnosis of esophageal perforation was still somewhat in doubt despite a slight elevation of temperature or even, occasionally, cervical emphysema. This type of case is variously described in the literature, and most authors have indicated that the patient recovers in a few days without surgical intervention. Head, Myerson, Van Eichen, Clerf and others have stressed the conservative management of this group of patients.

Careful watchful waiting means, of course, frequent temperature readings as well as repeated white blood cell counts. I thoroughly agree that any increase in findings indicates immediate operation. Dr. Pearlman mentioned laceration of the esophagus as opposed to perforation, and this difficult differential diagnosis is all important. Routine mediastinotomies in questionable cases would appear to be unnecessary, and I cannot agree wth Dr. Galloway that such an operation is simple and without danger.

This brings back the question of the term "prophylactic mediastinotomy." This term originally denoted drainage of the mediastinum through the neck in cases of external trauma of the esophagus, such as that from stabbing or a gunshot wound. At the present time the term is frequently used loosely to designate the opening of the retropharyngeal space and the placement of a gauze pack in the inferior part of the wound in an attempt to keep the infection from traveling into the mediastinum. Dr. Lederer and Dr. Fishman suggested clarification of this term in an article written in 1934 and gave reasons for and against the operation. Certainly the term should not be used when a simple mediastinotomy is done to drain an advancing mediastinal infection due to an esophageal perforation.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION OF OTOLARYNGOLOGY, AND NEW YORK ACADEMY OF MEDICINE, SECTION OF OTOLARYNGOLOGY

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Joint Meeting, Philadelphia, March 19, 1941

Some Interrelationships of Modern Hematology and Otolaryngology. DR. THOMAS FITZ-HUGH JR.

A brief review is presented of the more important hematologic disorders affecting the nose, oropharynx, larynx and auditory apparatus. Lingual mucosal strophulous glossitis and dysphagia in connection with anemia respond favorably to treatment directed toward the special type of anemia and the underlying cause. In the Plummer-Vinson syndrome curious veils of mucous membrane partly obstruct the lower part of the pharynx. Here, too, treatment directed to the disease is more effective than esophageal instrumentation. Aplastic anemia is frequently associated with oropharyngeal ulceration, necrosis and bleeding, and any severe anemia may produce vertigo, tinnitus and even deafness.

In leukemia, lymphosarcoma and Hodgkin's disease, not only the oral mucosa but also the tonsils and cervical nodes may present ulcerative and necrotic lesions. Epistaxis and gingival bleeding may occur, and in cases of mediastinal involvement recurrent laryngeal nerve palsy has been observed. Tooth extractions from patients with these diseases should be avoided because of the danger of hemorrhage.

In polycythemia vera there is visible cyanosis of the mucosae, with epistaxis, gingivitis and slight bleeding of the gums. Thrombosis of the blood supply of the inner ear may produce Ménière's syndrome. Patients with these conditions are bad surgical risks owing to the danger of postoperative thrombosis and hemorrhage.

In hemorrhagic blood dyscrasia, epistaxis, oropharyngeal hemorrhage and local purpuric lesions are common manifestations. Systemic hemostatic agents are not dependable. For the patients with thrombocytopenia tonsillectomy is considered a dangerous and dubious procedure and should not be undertaken until the disease has been brought under control, if necessary, by splenectomy. The most common drug causes of thrombocytopenia are benzene, allylisopropylacetylcarbamide (sedormid) and, less frequently, sulfanilamide or one of its derivatives.

Hereditary hemorrhagic telangiectasia may occur in the nasal, labial, oropharyngeal and lingual mucosae and on the tympanum; the only effective treatment is local coagulation or sclerosing applications or injections in the areas involved. The disorder is transmitted as a mendelian dominant, but atavism is not uncommon.

Acute infectious mononucleosis may simulate acute leukemia with its pharyngitis, stomatitis, generalized adenopathy, splenomegaly and lymphatic leukocytosis. The condition is self limited and probably due to a virus or toxoplasma which confers lasting immunity. Differential diagnostic criteria are discussed.

Agranulocytic angina is now believed due to a specific allergic shocklike mechanism of leukocytes, endothelium and bone marrow, the most common allergen being aminopyrine (dimethylaminophenyldimethylpyrazolon U. S. P.). A few cases have likewise been reported in which this angina occurred after ingestion of sulfanilamide and its derivatives. Here the best treatment is prevention. I am of the opinion that aminopyrine should not be prescribed, and sulfanilamide and its derivatives, like arsphenamine, should be administered only when indicated. In discussing all the aforementioned conditions, brief reference is made to the accepted form of treatment for the disease rather than to that for the local lesions.

Regarding the routine blood coagulation tests made before tonsillectomy in most hospital clinics, I suggest that for real protection against post-tonsillectomy hemorrhage there should be a test not only for clotting time but also for bleeding time, a tourniquet test, a complete blood count, a hemoglobin estimation, a platelet count and thrombogenic time determination. Most of the bleeding after tonsillectomy or adenoidectomy I have seen has been due to difficulties of hemostasis rather than to unrecognized blood dyscrasia.

DISCUSSION

DR. ANDREW A. EGGSTON, New York: The atrophic changes of the oral and nasal mucosa in anemia are of particular interest. There is not much information available as to just what histologic changes occur in the mucosa and seromucous glands or in the circulation of the oronasopharynx in severe anemia. More histologic data about avitaminosis with lesions in the upper respiratory tract would also be welcomed. It would aid in the evaluation of the vitamin therapy of many conditions affecting this region. The use of the various commercial vitamins in combating respiratory diseases has been freely and even loosely recommended; there is little scientific basis in man to support the claims made.

It is likewise important and sometimes essential for the differentiation of the anemias and leukemias to have the information that can be obtained from studies of the bone marrow. Either sternal marrow puncture or biopsy may be done. The marrow picture is particularly important if one is to determine whether aleukemic leukemia is present.

I have observed the initial lesions of leukemia in hypertrophied tonsil or pharyngeal lymphoid tissue. I recall a child from whom unusually large tonsils were removed; within six weeks they had recurred to a size equal to the original.

A blood count revealed that the patient had lymphatic leukemia. The other lymphoid structures of the neck and body were little involved. The mistaken diagnosis would not have occurred if leukemia had been suspected when the original and greatly enlarged tonsils were observed.

In several cases of leukemia, my associates and I have observed ulcerative and bleeding lesions in the oronasopharynx. All know the bizarre types of lymphosarcoma that involve the nose and throat. I have particular reference to the lymphoepithelioma or transitional cell lymphosarcoma, a common type of malignant growth in this region; it is of particular interest in that the primary tumor may be imperceptible and may be overlooked in the most thorough examination. With these tumors the diagnosis has only too often been made when secondary metastasis occurred in the neck and reexamination and biopsy demonstrated the primary tumor located in the tonsils or nasopharynx.

In regard to Hodgkin's disease, the diagnosis is usually easily made at biopsy. I recently encountered several patients with this disease for whom the diagnosis was made early from biopsy; they survived seven to ten years with proper irradiation of the involved tissues. I just wish to strike here this encouraging note—if the diagnosis is made early.

Dr. R. E. Buckley and I recently had a patient with chronic myeloid leukemia who had a most refractory purpura of the mouth and epistaxis. The exact cause of these symptoms was revealed only after examination of the blood. I recall another patient, a 62 year old woman with general purpura and considerable bleeding of the gums and oropharyngeal purpura and bleeding ulcers with extensive leukopenia from continued use of sodium amytal. In my experience the incidence of these milder forms of leukopenia and that of true agranulocytic angina have certainly decreased since it has been shown that they are usually due to an allergy for certain drugs, chief of which are those of the aminopyrine group. In cases of hemorrhage prothrombin time determination has a real place in proper management. This test is a guide as to the need for vitamin K and has been of real value in cases of epistaxis and ocular hemorrhages. The test is a bit difficult to interpret and should be done only by one who has had extensive experience with its technic.

In recent years more and more patients with infectious mononucleosis have come under observation. Many of these have been adults. Probably a better understanding of the mononuclear blood cells has been responsible for the better hematologic diagnosis of this condition. The heterophil antibody reaction has also been of great aid in confirming probable diagnoses. It has been my impression that few characteristic lesions occur in the nose and throat of the patient with this disease; the diagnosis is usually made on the basis of the swelling in the neck and the blood picture.

This is a digression, but I wish to emphasize the importance of a shift to the left of the blood count in the prognosis of progressing infection, also the aid that a sedimentation test offers in the diagnosis of chronic sinusitis, mastoiditis or abscess of the brain.

I wish to refer to the value of the Pels-Macht phytotoxic test in the differential diagnosis of pemphigus lesions of the oronasopharynx. This has been of real service to me on several occasions.

My associates and I have also observed severe diffuse ulcerative and hemorrhagic lesions in the pharynx of a young man, which were local manifestations of erythema multiforme. All laboratory tests were noncontributory in the diagnosis of this condition.

Dr. Fitz-Hugh has made it quite evident that a thorough history and physical examination and hematologic studies are essential to the diagnosis and treatment of atrophic ulceration and hemorrhagic lesions of the oronasopharynx.

Some External Nasal Deformities and Methods Used in Their Repair.
Dr. Warren B. Davis, Philadelphia.

Ill advised articles on plastic surgery in lay publications have unduly emphasized the repair of insignificant deformities of the nose. Such defects are not

infrequently exacerbated by attempted correction, and the result is dissatisfied patients. This situation should be combated more energetically by the organized groups of ethical plastic surgeons. In the present paper the various types of real nasal deformity and their treatment are discussed.

The best time for the correction of congenital deficiencies of nasal bones and nasofrontal processes is when the patient is 16 to 20 years of age. Earlier correction is followed by increasing disproportion between the size of the developing face and the size of the transplanted cartilage, which does not grow. The treatment of choice for such deficiencies is the transplantation of an autogenous piece of costal cartilage. Procedures contributing to successful results are prolonged preoperative massage, use of the curved costal cartilage chisel with the convex surface upward and insertion of the cartilage through a columellar incision. Nasal depressions of infectious, traumatic or operative origin are corrected rather similarly but may require a supporting cartilaginous post in the columella. The large perforations of the nasal septum in some syphilitic depressions may permit only partial correction.

A lateral deviation of the nose resulting from an old fracture without depression is corrected by partially dividing the frontonasal processes of the maxillas near their attachments to the bodies of the maxillas and fracturing the remaining attachments by digital pressure or by means of a mallet. In many cases submucous resection of a portion of the deflected septum affords a better respiratory area and prevents spring or back pressure in the septum. In cases of slight depression between the lower ends of the nasal bones and the nasal tip, the removed cartilage may be used to correct the deformity.

In excessive convexities of the dorsum with drooping of the tip of the nose, correction is made by freeing the overlying skin and subcutaneous tissue from the cartilaginous bridge, and the periosteum from the bone to be removed, through an incision made within the superior portion of the anterior part of the nasal passages. The use of rasps of assorted sizes and shapes is preferable in most instances. The importance of meticulous removal of bone dust and careful cleansing of rasps before reinsertion is stressed. For large humps a saw may be necessary.

For rhinophyma elliptic excisions are recommended. Two operations, approximately one month apart, are usually required. Serial applications of a saturated solution of trichloroacetic acid at certain intervals over several small areas at one time will tend to minimize undue thickening of the epidermis still remaining around some of the larger pores. Illustrative cases of chondroma, of defects due to surgical excisions for lesions suspected of being malignant or, to application of "cancer pastes," and of alterations caused by carcinoma and by heavy irradiation of tissues are described with the treatment used in each case and the results obtained.

Pigmented hairy nevus may be even more disfiguring than many actual deformities. A case in which treatment was by excisions and immediate skin grafting is described.

In planning plastic reconstruction, one should visualize the ultimate result desired and outline the various steps in repair. Knowing the number of contemplated operations and the probable intervals helps the patient in arranging adequate time for the operations and convalescence.

Accuracy in outlining flaps, gentleness in handling tissues and evenness in approximating skin margins all tend to minimize scar tissue and thereby assist in securing the best cosmetic results.

DISCUSSION

DR. ARTHUR PALMER, New York: The service of rhinoplasty in the field of mental hygiene has not been fully appreciated by physicians. Maintenance of a person's morale and efficiency depend on personal appearance as well as on physical fitness. In certain instances mental distress resulting in lack of confidence and a feeling of inferiority reduces the subject to a social, economic and psychologic level considerably below that to which his natural abilities entitle him. The ill effects of a deformity are often out of proportion to the disfigurement. This is

particularly true of the person with a traumatic lesion, since the victim compares his present unhappy state with his former happy state, whereas in the case of a congenital lesion there is no such comparison.

Dr. Davis has given a fully illustrated discourse on numerous types of external nasal deformities in a most instructive manner. Time does not permit any attempt on my part at a complete discussion of his paper. I shall confine my remarks to the correction of the so-called saddle back deformity of the nose.

While recently a number of plastic surgeons have employed preserved cartilage as material for transplantation with reasonably good results, I still believe that the patient's own cartilage—costal, septal or aural—is better. It is much less likely to become infected or to be absorbed. It should be free of perichondrium, should be of the correct size and shape, should preferably be introduced through a skin incision in the columella and should fit snugly.

In contrast to the marked local reaction which usually follows removal of a nasal hump with narrowing of the nose by mobilization of the bony walls, little reaction follows the transplantation of autogenous cartilage grafts, and the cartilage remains viable.

Poor cosmetic results may follow (1) use of too large a graft, (2) failure to strip off the perichondrium, with subsequent bowing of the transplant, and (3) migration of the graft because it does not fit snugly. With proper surgical technic, infection should play a decidedly minor role.

Pharmacology and Toxicology of Sulfanilamide and Its Derivatives. Dr. Harrison F. Flippin, Philadelphia.

The rational utilization of sulfanilamide and its derivatives demands, first, an understanding of their absorption, distribution and excretion and, second, early recognition of toxic manifestations. Owing to the absorption of these drugs from the intestinal tract into the blood stream within a few hours after oral ingestion and the rapid subsequent diminution of their concentration in the blood, it is important to administer a large initial dose, followed by smaller doses at four hour intervals, day and night, until complete clinical cure is obtained.

After absorption into the blood stream, these drugs are split by the liver into a free or active factor and an acetyl or inactive factor, which is more toxic. The diffusion of these drugs into body tissues and fluids varies in relation to vascular supply, so that in areas of chronic infection, diffusion in bone and necrotic tissue may be deficient. With the exception of sulfathiazole (2-[paraaminobenzenesulfonamide]-thiazole), they pass readily into a cerebrospinal fluid and the red blood cells. They are excreted mostly in the urine, both as free and as acetylated compounds, and, with the exception of sulfadiazine (2-sulfanilamidopyrimidine), within twenty-four hours. There is considerable reabsorption by the tubules, and elimination of these substances is reduced in the presence of diminished excretion of nitrogen. It is extremely important to facilitate excretion of the acetyl compounds by the kidney by maintaining an adequate urinary output of at least 1,200 cc. daily, which is achieved by forcing the consumption of fluids, either orally or, if necessary, parenterally.

The most important factor influencing the incidence and severity of toxic manifestations is the length of time the drugs are administered; most severe reactions develop after the twelfth day of therapy. Children, Negroes and well nourished persons with normal renal function seem to tolerate these drugs better than other subjects. The incidence of certain toxic effects depends on whether the patient is ambulatory or confined to bed. Except men with gonorrhea, patients receiving this treatment should be kept in bed. The dizziness complained of by ambulatory patients is significant, especially if the patient drives an automobile. The most frequent toxic reactions from these drugs are nausea and vomiting, usually appearing during the first twenty-four hours of treatment. Vomiting is mild in patients treated with sulfanilamide, sulfathiazole and sulfadiazine, but after the administra-

tion of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) it constitutes a serious problem and may necessitate discontinuance of treatment. A careful check on fluid and salt balance is indicated by severe vomiting.

Cyanosis is often observed but may be disregarded. Drug fever, frequently followed by dermatitis, hemolytic anemia or neutropenia is an indication to stop treatment. Drug rashes may occur at any time, especially after the fifth day of treatment; if treatment is not stopped, extra precautions must be taken. If psychoses develop after the infectious process is under control, the drug should be discontinued. Conjunctivitis has been seen only after administration of sulfathiazole; hematuria develops after the use of all drugs except sulfanilamide in a small number of cases. Unless a considerable number of red blood cells are detected or the ureters become obstructed, cautious treatment may be continued, but it should be kept in mind that severe renal insufficiency may follow. When renal calculus develops, treatment must be stopped. The mild hemolytic anemia that sometimes appears is not considered serious. Hepatitis, purpura haemorrhagica and neuritis rarely occur with sulfanilamide or sulfapyridine therapy.

DISCUSSION

DR. WESLEY C. BOWERS, New York: Dr. Flippin's paper covers very thoroughly the pharmacology and toxicology of sulfanilamide and its derivatives.

He speaks of the importance of adequate dosage in obtaining good therapeutic results. This is a point which cannot be overemphasized. I have in mind the case of a 20 pound baby (9,071 Gm.) with meningitis following mastoiditis. Forty grains (2.59 Gm.) was given daily without improvement. The amount was increased gradually to 90 grains (5.82 Gm.) daily, and this dosage was continued for at least ten days without toxic symptoms; the clinical picture cleared up, but the use of the drug was continued for another ten days, in diminishing quantities.

Many doctors are in the habit of prescribing two to three tablets daily without checking the blood or urine. Sooner or later such a procedure will bring about bad results, for which the drug, not the dosage, will be unjustly blamed.

As Dr. Flippin has well said, "If the patient is sick enough for chemotherapy, he is sick enough to be in bed."

The administration of the drug should not be stopped with the disappearance of clinical symptoms but should be continued for some days longer; the length of time depends on the severity of the condition.

As the diffusion of the drug depends on vascularity, it is important to start medication early, before necrosis has developed; necrotic areas, with their poor blood supply, will not respond to chemotherapy but will require drainage. This explains the masking effect of chemotherapy in acute mastoiditis. The vascular middle ear clears up; the necrotic area in the mastoid process does not. Hence, when administration of the drug is stopped, the mastoiditis recurs with increased severity. For this reason, I believe that chemotherapy without operation is not justified in cases presenting a classic clinical picture of mastoiditis.

As a result of my experience in the treatment of 793 patients with acute purulent otitis media in the past three years, chemotherapy having been used in half of them, I have arrived at the following conclusions:

1. If chemotherapy is given early, before bone destruction occurs, the duration of discharge and the number of mastoidectomies is diminished by about 50 per cent.

2. When the clinical picture strongly suggests mastoidectomy, it is safer to operate.

3. After uncomplicated mastoidectomy, it is better not to give sulfanilamide or a derivative.

4. Complicated mastoiditis requires intensive chemotherapy.

5. At times it is necessary to stop medication in order to obtain the true picture, as the drug cures the middle ear while progressive bone destruction is taking place in the mastoid process.

Sulfapyridine often produces severe toxic symptoms, sometimes simulating meningeal or labyrinthine involvement so closely as to be alarming. For this reason, sulfathiazole or sulfadiazine are to be preferred in infections of the ear, nose and throat, except in cases complicated by meningitis. Dr. Flippin stated that he has not seen gross hematuria from sulfathiazole; I have seen 1 patient with extremely severe hematuria following treatment with that drug.

When the use of chemotherapy is producing unsatisfactory results, changing to another form of the drug or to serums to stimulate the antibacterial antibodies will often bring a better response.

Chemotherapy has unquestionably added greatly to the confidence of physicians in their ability to conquer acute otitis media, mastoiditis and fulminating sinusitis. They have always been apprehensive of meningitis as long as the ears and sinuses continued to discharge. During the past three years, I have had 5 patients with meningitis, 4 of whom recovered.

One should be ever mindful that chemotherapy used in ear and nose infections may so obscure the clinical picture that there will be increased danger of the unheralded sudden onset of grave complications, such as meningitis and osteomyelitis, and that under the influence of these drugs all the symptoms of mastoiditis and sinusitis may disappear while the lesion is actually spreading.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
President-Elect: Dr. Ralph I. Lloyd, 14-8th Ave., Brooklyn.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-24, 1941.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
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American Laryngological Association

President: Dr. T. E. Carmody, 227-16th St., Denver, Colo.
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American Otological Society

President: Dr E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
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